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HEART DISEASE



CHOREA IN A CHILD EIGHTEEN MONTHS OLD.

The photographs were taken in succession, and show the bizarre attitudes assumed in chorea, particularly the facial expression.

CLINICAL PEDIATRICS

HEART DISEASE IN INFANCY *and* CHILDHOOD

BY

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WITH A CHAPTER ON ELECTROCARDIOGRAPHY

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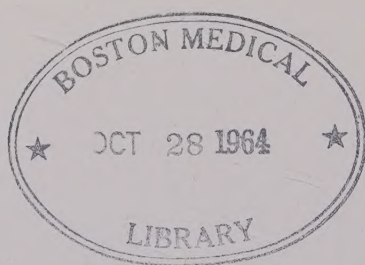
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PREFACE

In writing this book, our attempt has been to present the problem of heart disease in childhood as simply and as accurately as possible. No excuse is offered for the fact that most of the text is devoted to rheumatic fever, since it is chiefly rheumatic heart disease which is met in children. We have tried to show clearly that rheumatic fever is not simply a disease of the joints, the heart or the brain, but that it is an infection which is widespread in its effect on the organism. We hope, also, that some who read the book will be led to think of rheumatic fever in terms of a tremendous public health problem which has possibilities of solution.

Many of the questions as to the etiology, the pathology and the pathological physiology are not yet clear. We have tried to avoid controversial subjects, and to present only what is known to be true, or what is probably true in the light of present knowledge.

The bibliography appended includes but a small part of the literature on rheumatic fever and on problems relating to this disease. Some of the articles referred to have extensive bibliographies, such as Sacks' review of the pathology of rheumatic fever; Paul's most interesting article on the pleural and pulmonary lesions, and Swift's recent paper on rheumatic fever. Anyone wishing to go more deeply into any of the aspects of the disease can get an idea of where to look by referring to the bibliography in this book.

We are glad to express here our deep appreciation of the help given us by Dr. Homer F. Swift, in lending us illustrations and in criticizing the section dealing with the specific etiology of rheumatic fever; and by Dr. Leon T. LeWald who allowed us to use two of his photographs and who also read and criticized the part of the book which deals with x-ray examination of the heart. Dr. Douglas Symmers, Director of the Pathological Laboratory of Bellevue Hospital, very kindly allowed us to use photographs of specimens in his laboratory, and Dr. Clarence de La Chapelle was particularly courteous in helping

us with the illustrations of the pathology of rheumatic and congenital hearts. Thanks are also due Dr. Alexander Lambert for the use of electrocardiograms from his laboratory; and to all others who have helped us.

CHARLES HENDÉE SMITH
LUCY PORTER SUTTON

PUBLISHERS' ANNOUNCEMENT

The publishers take pleasure in presenting to the medical profession the series of monographs of which this volume forms a unit.

The many inquiries which reached them proved, in advance of publication, that the work should be in monographic form and clinical in its presentation.

The series when completed will, they believe, be the most useful for the audience for whom it is written, the general practitioner of medicine, that has been presented in its particular field.

The authors are all men of wide experience and, in the main, teachers. The combination makes the work authoritative and of the utmost service in a field which has been often termed a "therapeutic specialty."

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HEART DISEASE

HEART DISEASE IN INFANCY AND CHILDHOOD

CHAPTER I

INTRODUCTION

One has to work in a children's cardiac clinic or hospital only a short time to be strongly impressed by the size of the problem of heart disease in childhood. The child who is limited in his work and play from this cause presents a pathetic picture. After seeing the hundreds of children who go through an active clinic, the question soon arises, "What can we do about it?" We hear of the "rising tide of heart disease," and for those who are primarily interested in the health of children we must determine as nearly as we can how far this tide includes children. To do this and to get an idea of the problem in the United States as a whole we must go to the vital statistics.

We find that there is a rapidly rising tide, but that, although heart disease is the leading cause of death in the United States Registration Area, the greatest number of the deaths from heart disease occur in late middle life and in old age. This is probably due largely to the fact that expectation of life has increased through the control of infectious diseases, leaving the organism to succumb to the senescent cardiovascular diseases. Heart disease may be roughly divided into three groups by causes, namely, (1) rheumatic, (2) syphilitic, and (3) degenerative and senescent.

Deaths from heart disease aside from the congenital abnormalities below the age of forty are almost entirely rheumatic, and practically never from either of the other two causes. The deaths at the ages of forty to sixty are attributed to syphilitic heart disease, and after sixty to senescent and degenerative causes. Thus rheumatic fever contracted in childhood is reflected in the cardiac deaths up to the age of forty, which comprise 25 per cent of all cardiac deaths. These figures are sufficient to show that heart disease in childhood, and its chief cause, rheumatic fever, constitute a tremendous public health problem. A still further analysis by communities can be made. Among school children in the United States Registration Area as a whole, heart disease is the fourth among the causes of death. In New York City

the fact has been brought out that among schoolgirls it is the leading cause of death, and among schoolboys it is second only to accidents. Among young adults in New York City between fifteen to nineteen years of age it is the second cause of death in both sexes. This analysis points to heart disease in childhood as a problem more important to urban than to rural communities.

Since neither heart disease nor rheumatic fever are reportable conditions we have no figures which show the morbidity rate. Various estimates, however, have been made. Emerson, for instance, says that heart disease interferes with the work, play or comfort of at least two million individuals in the United States, and that at least fifteen out of every one thousand school children have heart disease. This means fifteen thousand children in a city the size of New York.

During the past fifty years while the number of deaths from tuberculosis has declined 44 per cent, the number from heart disease has increased 42 per cent. Emerson also states that for every death from heart disease there are probably seventeen people suffering from it, while there are only seven cases of tuberculosis for every death from that cause. Dublin gives the more conservative figures of ten cases of heart disease for every death from this cause.

A realization of the size and importance of this problem came into the minds of a group of physicians almost simultaneously in the years 1912 to 1916. Guile, in 1912, started an evening adult class at Bellevue Hospital for cardiacs who were working. In 1915, St. Lawrence started a children's cardiac class at St. Luke's Hospital. In 1916, Smith organized at Bellevue Hospital the class for children which has been endowed by a fund in memory of the late John H. Huddleston who was working on a similar plan at the time of his death. The idea spread rapidly and in a few years there were twenty or more organized classes. The Association for the Prevention and Care of Heart Disease was organized by Conner, Hart, Emerson, Halsey and others.

In four boroughs in New York City, forty-six cardiac clinics were in operation during 1928. Of these, twenty-one cared for children only, and had an enrollment of about five thousand cases. This does not represent every case in New York City by any means, as some are cared for by their own physicians and many go unrecognized for years. The Huddleston Memorial Cardiac Class at Bellevue Hospital, which was organized in 1916, has treated two thousand cases, and there are now over four hundred on the active list, that is, four

hundred children who report to the clinic at fairly regular intervals. Of these, 60 per cent are children who have fully developed heart disease. Thirty per cent are patients who show abnormal signs or symptoms not definitely proved as yet to be caused by organic heart lesion. Ten per cent are children who have had one or more of the diseases known frequently to be associated with heart disease, the so-called "potential cardiacs." The number of children enrolled as potential cardiacs in the various clinics is low, in some less than 5 per cent, indicating that more attention is being paid to those children who have heart disease in various stages, and who will probably die before they reach the age of forty, than to the "potentials," whose health and lives might be saved by the right kind of care. The cardiacs need care, but the best we can do for many of them in the light of our present knowledge is to prolong life by a few years, too often a life of invalidism.

Emphasis on the prevention of rheumatic fever and its ensuing heart disease is a more constructive point of view. On the other hand, proper care of real cardiacs often gives remarkable and unexpectedly good results. Comparison of figures from heart disease and tuberculosis, about the prevention and cure of which so much progress has been made, shows from another angle the widespread importance of the problem of heart disease. Dublin has estimated that at ten years of age an individual is three times as likely to die from heart disease as from tuberculosis, at thirty a man is four times as likely to die from heart disease as from tuberculosis, and a woman at thirty is five times as likely.

Since many of the cardiac deaths in adults are caused by heart disease contracted in childhood, the need of preventive work in the early years is clear. Approximately 25 per cent of the cases of heart disease are caused by rheumatic infection. The death-rate from this cause declined under the age of five years in the period of 1900 to 1914, while it remained unchanged between the ages of five to forty-five. Swift has published a graph showing the decline of the death-rate for acute rheumatic fever from 1900 to 1921, but there has not been a corresponding decline of the death-rate for this same period from heart disease. Several factors are possibly influential in the declining death-rate from rheumatic fever. One is the better care of the patient who has the disease, particularly the widespread use of salicylates in large doses, which prevents the hyperpyrexia which formerly formed so large a part of the picture of acute rheumatic

fever in adults. More accurate diagnosis is made possible by recognition of the fact that the heart may be, and usually is to some extent, acutely involved during the attack of rheumatic fever, so that death is then given as acute endocarditis rather than acute rheumatic fever. Possibly too, though in the absence of statistics this cannot be proved one way or the other, there is an actual decline in the incidence of the disease.

The figures quoted give a general and impersonal idea of the extent of the problem of heart disease in childhood, but a personal realization of the question is borne in upon one only from working in intimate contact with patients. Rheumatic fever is probably preventable to a degree, to how great a degree we do not yet know. Until we are able to obtain definite figures on the incidence of this disease we will not really know how we stand in regard to its increase or decrease. The feeling is growing that it is communicable, perhaps in very much the same way as tuberculosis, and probably some day it will become a reportable disease. When this occurs the advance towards prevention will go forward more rapidly, and we shall be able to control to a certain extent the number of children who develop heart disease.

The study of heart disease in childhood may be divided into several groups, the first and most important of which is rheumatic heart disease. In this group are included (1) the children who have had acute rheumatic fever, chorea, "growing pains," carditis, but have not yet developed permanent organic lesions of the heart—termed "potential cardiacs"; (2) those who already have well-developed organic lesions of the heart; and (3) children who present abnormal physical signs, or symptoms without physical signs, which cannot be definitely ascribed to organic heart disease. The aim with the first class is to prevent the development of heart disease if possible. The problem with the second group is to keep these children in as good general condition as possible in the hope of prolonging life, or of preventing further involvement of the heart and further attacks of rheumatic fever. With the others the chief consideration is the diagnosis, either to establish the fact of the presence of heart disease or to rule it out definitely. This is frequently an extremely difficult task, and often only prolonged observation over a period of years will answer the question satisfactorily, if at all.

Another large group is composed of the children who have congenital malformations of the heart. They are interesting and im-

portant chiefly as presenting anatomical curiosities. Little if anything can be done as to prevention or for the heart itself. But the child needs care just as much as a patient with acquired heart disease. It is necessary to be familiar with the various forms of congenital heart disease, because of the importance of correct diagnosis. It is not always easy to differentiate between an acquired organic lesion and a congenital lesion. Furthermore, a child who has a congenital lesion may even have a rheumatic lesion superimposed on the congenital condition. In such cases the acquired lesion may be deemed the more important in the handling of the child.

Lastly, the functional disorders of the heart must be considered. These do not hold the important place that they do among adults, but they do occur and must be recognized and treated when present.

The major portion of this volume will be concerned with the diagnosis and treatment of rheumatic heart disease, and of the various rheumatic infections. For a true presentation it is necessary to appreciate the fact that heart disease cannot be considered purely in the light of structural changes which have occurred in the heart, or the mechanical and functional disabilities which ensue; but that the heart is only one of several organs in the body which may be attacked by the rheumatic microorganism. It is not accurate to say that chorea or acute rheumatic polyarthritis cause heart disease. A truer conception is that rheumatic fever is a disease which may attack the brain, the joints, the heart or other structures, either together, separately, or successively. In essence, the problem of heart disease in childhood is the problem of rheumatic fever in childhood.

CHAPTER II

ETIOLOGY OF RHEUMATIC HEART DISEASE

Accurate knowledge of the natural history of any disease is vital to a true conception of it, its spread, modes of transmission, entrance to the organism, and methods of attack. At present, there are great gaps in our knowledge of rheumatic fever, but facts are gradually being accumulated, and in time we shall certainly know as much of it as we do of other diseases. For the time being, what we do know is more suggestive than definite, and what facts we know will be presented for what they are worth.

Race and Climate.—Among certain races, for instance the natives of India, China and Japan, heart disease appears to be rare, while in certain countries, namely Great Britain, North America, Germany and the Scandinavian countries, the death-rates from heart disease are high. This suggests strongly the possibility that climate is a factor in the incidence of heart disease, but the available data are not conclusive. Among English people living in the British colonies the rate is low. One suggested explanation for this is that their children are sent back to England at the age periods when rheumatic infections are most likely to occur. Dublin points out that there is a great difference among the nationalities which go to make up the population of the United States. The Irish in this country have a high heart-disease rate, but this is not true for the Irish in Ireland. Colored people in this country have been said to be relatively free from rheumatic infection, but colored children with rheumatic polyarthritis, chorea or heart disease are not uncommon at Bellevue Hospital. The negro population of Manhattan has increased rapidly in recent years and it may be that the fact that negroes formerly resided chiefly in the southern states in childhood accounts for their apparent exemption from rheumatic infection. In fact, there is a variation in the incidence of heart disease according to sections of this country, which has such great climatic differences. The southern mountain states show a lower incidence of heart disease (that is, the death-rate is lower) than the Pacific, Middle Atlantic and New England states.

This variation according to locality holds true for the death-rate from acute rheumatic fever also.

Racial susceptibility to heart disease is apparently a factor, although here, too, our data do not allow us to draw final conclusions. If we find that a certain racial stock in this country has a high incidence but is relatively free from the disease in its own country, we can deduce that climate is more of a factor than race.

Table I shows an analysis by race of 511 children in the general children's clinic compared with 506 children at the cardiac clinic at Bellevue Hospital. This shows that, although the Italians form only 19.56 per cent of the population of the general clinic, they comprise 23.5 per cent of the cardiacs. Similarly, 27.4 per cent of the children in the general clinic are Jewish, while only 21.94 per cent of the cardiacs are of this race. This suggests that the Italians of New York City are somewhat more susceptible to rheumatic heart disease than are the Jews. A more striking comparison is shown in the figures for the Irish. Of the cardiacs, 15.8 per cent are Irish, although only 4.3 per cent of the patients of the general clinic are of this race.

TABLE I*
RACE INCIDENCE

Race	Per Cent in Cardiac Clinic	Per Cent in General Pedi- atric Clinic	Race
Italian	23.5	27.4	 Jewish
Jewish	21.94	19.56	 Italian
American	19.0	14.48	 American
Irish	15.8	5.47	 Armenian
German	5.33	5.09	 Spanish
Polish	3.95	4.7	 Austrian
Hungarian	2.76	4.3	 Irish
Austrian	1.58	4.1	 German
English	1.38	3.7	 Polish
Spanish	1.18	3.3	 Greek
Greek	.98	2.34	 Rumanian
Czechoslovakian	.98	1.6	 Czechoslovakian
Colored	.59	1.6	 Colored
Finn	.19	1.17	 Hungarian
Ukrainian	.19		

* From L. P. Sutton, *Am. Heart J.*, St. Louis, 1928.

Although there are no English among the sample from the general clinic, 1.38 per cent of the cardiacs are English. The type of population of the neighborhood in which a clinic is located may, of course, affect the races which attend the clinic, but Bellevue draws

its patients from all parts of the city and even from some of the suburbs, so that this factor does not operate as it would in a strictly neighborhood clinic.

Such figures as have been given, merely suggest that certain facts may be true, and only the accumulation of data from very large samples of the population will give an accurate idea of the effect of race and climate on the incidence of heart disease.

Season.—Ingerman and Wilson, in their study of 185 cases, found that 31 per cent of the rheumatic manifestations occurred during the winter months, 33 per cent in the spring, 29 per cent in the summer, and only 7 per cent in the fall. The figures from 584 ambulatory and hospital patients at Bellevue (Chart I, Table II) do not coincide with these exactly, particularly in the fall group, which is more than twice as high for the Bellevue sample as for the cases studied by Ingerman and Wilson. April and May stand out in the Bellevue study as having the greatest number of cases of chorea and acute rheumatic fever.

TABLE II
SEASONAL FLUX OF RHEUMATIC INFECTIONS: PRIMARY DIAGNOSIS CHOREA OR
ACUTE RHEUMATIC POLYARTHRITIS

195 Clinic Patients
131 In-Patients

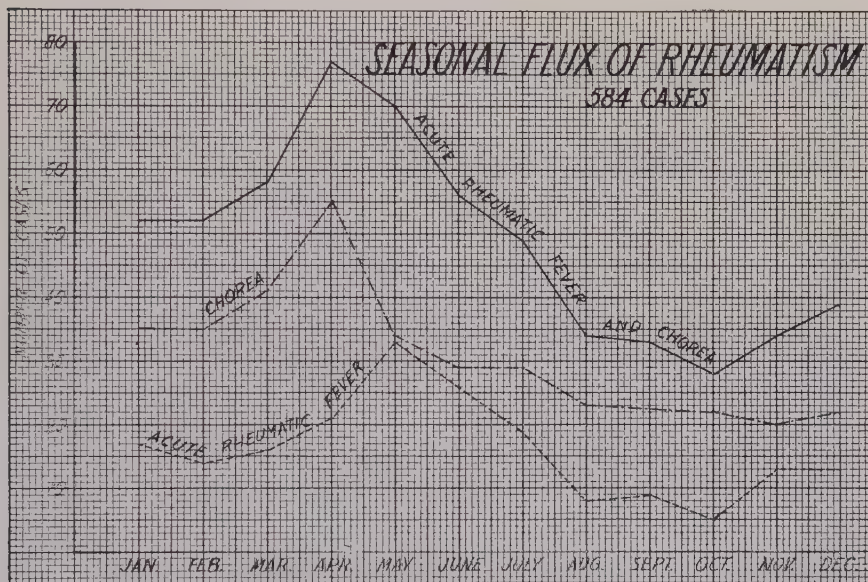
Month	Number of Cases	Per Cent
January	30	9.2
February	29	8.89
March	30	9.2
April	44	13.49
May	39	11.96
June	28	8.58
July	30	9.2
August	23	7.0
September	17	5.2
October	17	5.2
November	20	6.1
December	19	5.8

This differs from the seasonal variations of acute rheumatic fever and chorea in London hospitals which appear to be quite regular, with little variation in different years. There is a rapid rise in late summer, a high incidence in the fall, and a sudden drop at the beginning of the winter. Climatic conditions may be a factor which accounts for the difference in the figures of New York and London. Young's figures indicate that excessive rainfall has a predominating influence

on the prevalence of rheumatism, but we were unable to find any correlation between precipitation and temperature, and the incidence of rheumatic fever.

Coombs attempted to determine the seasonal incidence of rheumatic carditis aside from other forms of rheumatic fever, and found that the incidence of fatal rheumatic carditis in London was maximal in December and January and minimal in July and September. There is some difference in the seasonal incidence in New York in chorea and

CHART I
SEASONAL FLUX OF RHEUMATISM*



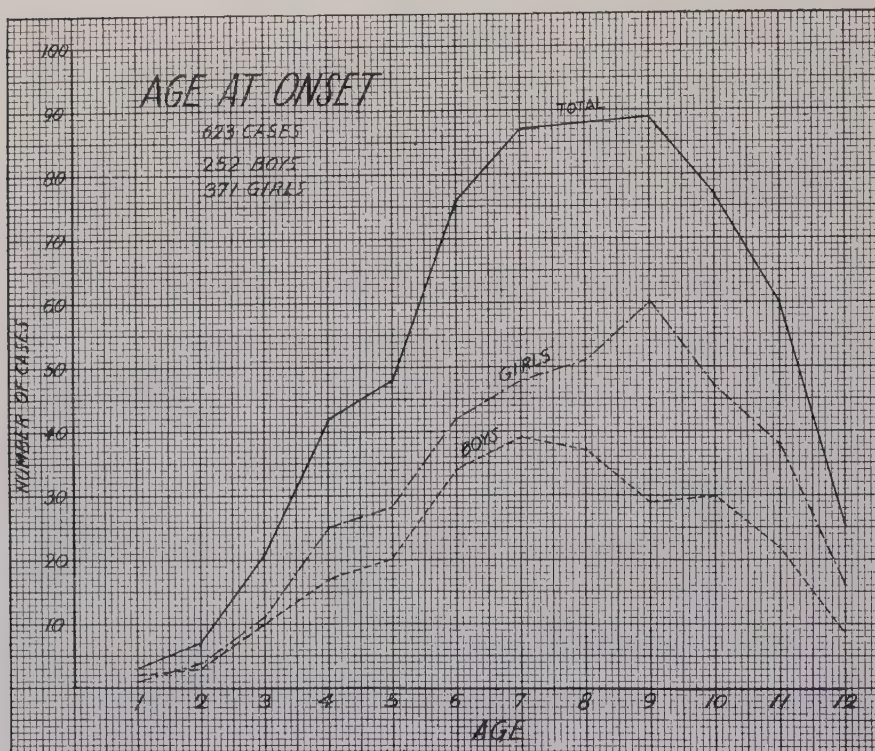
* From L. P. Sutton, *Am. Heart J.*, St. Louis, 1928.

acute rheumatic fever, the peak for the bedridden cases of chorea coming in April, that for acute rheumatic polyarthrititis in May (Chart I). This also differs from the English findings, where the peak for chorea is January and September, for acute rheumatic polyarthrititis February and November.

Age.—The age at the onset of a rheumatic infection is not always easy to determine accurately. Where the disease starts as a clear-cut chorea or acute rheumatic polyarthrititis, the date of onset is obvious, but too often the symptoms escape the notice of the parent. There may be no symptoms sufficiently acute to call attention to the carditis in its early stage and the memories of clinic patients are notoriously

inaccurate. However, numerous attempts have been made to determine the ages at which the incidence of rheumatic infection is greatest, and the various series agree fairly well. The peak of Ingerman and Wilson's curve came at five years, Miller and Poynton give seven years and our own figures show little difference in the ages, six, seven, eight, nine, and ten (Chart II), at which ages 67 per cent of the cases

CHART II
AGE AT ONSET *



* From L. P. Sutton, *Am. Heart J.*, St. Louis, 1928.

were first attacked. Our figures include no patients above the age of twelve.

The age at which the incidence was maximum was nine. These figures seem to show very clearly that it is during the first decade that the organism is most susceptible to rheumatic infection. Coombs found that the onset in three-fourths of over six hundred cases occurred before the age of fifteen, that two-thirds took place between the ages of five and fifteen, 15 per cent from fifteen to twenty-five years, and only 10 per cent over twenty-five. It is generally thought

that infection before the age of three is rare, and Coombs states that among his clinic patients only 7.1 per cent showed active infection during the first five years of life.

The Bellevue figures show 17 per cent of the children to have been infected during the years two, three, four, and five, of which 3.5 per cent are in the years two and three. Denzer reported three cases in children, twenty, twenty-three, and twenty-four months old, respectively, who died of rheumatic endocarditis. In two, typical Aschoff bodies were found at necropsy, and in the other which was not autopsied, characteristic subcutaneous rheumatic nodules were found. We have had one case of endocarditis at eighteen months, and one of acute chorea at the same age. Schroeder reported a case of chorea associated with endocarditis and myocarditis, in an infant of twenty months, in whom Aschoff bodies were found at autopsy. Denzer feels that any infant dying of cardiac failure of uncertain origin should be searched carefully at autopsy for the lesions of rheumatic origin. If we bear in mind that rheumatic fever may and frequently does attack the heart itself primarily, more cases of rheumatic carditis will probably be diagnosed at earlier ages.

Sex.—Females appear to be somewhat more frequently attacked than males. Of 506 children at the Bellevue clinic with organic heart disease, 214 were boys and 292 were girls. The combined percentages of Bellevue and another smaller clinic were analyzed by sex and it was found that the peak for the boys occurred two years earlier than it did for the girls (Chart II). In other words, the greatest number of the boys were affected at the age of six years, while the greatest number of girls were first attacked at nine years. With this may be correlated another finding, namely, that among 253 girls with rheumatic heart disease, 20.94 per cent gave no history of any rheumatic infection prior to the discovery of heart disease, while among 174 boys, 14.36 per cent gave no history. Both sexes show 18.26 per cent with no rheumatic history prior to the onset of heart disease.

This suggests that girls may be more susceptible to rheumatic carditis than boys, since this is the rheumatic manifestation which is most insidious in its onset and therefore most likely to be absent in the history.

Heredity.—*Family Incidence.*—There is little evidence of the possibility that the virus of rheumatic fever may be passed from mother to fetus, although it is quite possible that some inherent susceptibility of the organism may be transmitted. The familial factor,

however, is striking. St. Lawrence was the first to study this extensively and he reported the incidence of rheumatic heart disease in a series of one hundred families of which one or more members of the family were enrolled in the children's cardiac clinic at St. Luke's Hospital in New York. He found an incidence of 50 per cent, that is, if one member of the family has a rheumatic infection, there is one chance in two that at least one other member of the family will be found to be affected.

A recent report published by the Medical Research Council of England on *Social Conditions and Acute Rheumatism*, based on a very careful survey of 721 families containing at least one child who was at the time, or had been under treatment for rheumatic infection, states that there is a high family incidence, up to 40 per cent. This family incidence seems to be slightly higher for rheumatic fever than for tuberculosis (Table III).

TABLE III
FAMILY INCIDENCE OF RHEUMATISM *

NUMBER OF MEMBERS OF SAME FAMILY AFFECTED	SOURCE OF DATA				
	C.H.G.O.S.	R.H.S.C.G.	S.T.H.	St. Law- rence	Faulkner and White
2	33	47	100	33	—
3	5	11	44	14	—
4	3	—	11	2	—
5	—	—	6	1	—
2 or more affected.....	41	58	161	50	71
Per cent of families.....	39 ± 4.3	29 ± 3.2	40.3 ± 2.5	50 ± 5	35.5 ± 3.4
Number of families....	121	200	400	100	200

* From *Social Conditions and Acute Rheumatism*, London, 1927; by permission of the controller of H. M. Stationery Office.

Environment.—A striking feature of rheumatic heart disease is that it is rarely seen among private patients, but is common among clinic and hospital patients. A comprehensive study made to determine, if possible, the factors which determine the incidence of rheumatism in childhood was undertaken by the Medical Research Council of England. The special aim was to see whether there was any evidence of contagion in rheumatism. The word “rheumatism” is used in the report of the investigation to denote rheumatic polyarthrititis, chorea, joint pains, associated with heart disease or chorea. The following general conclusions were drawn by G. F. Still:

"It seems clear that apart from any specific cause of rheumatism there are factors entering into the causation of rheumatism which belong rather to environment. Strong evidence of the existence of factors of this kind is afforded by the social incidence of rheumatism—its incidence is chiefly on the poorer classes—721 children attending hospitals in London and Glasgow were examined, and their family histories and environment investigated. For purposes of comparison a similar investigation was made regarding 200 children attending hospitals for disease other than rheumatism, and also with regard to 1872 children residing in Poor Law Institutions. There was found to be clear evidence of family incidence. Rheumatism was common in the parents or brothers and sisters of the rheumatic child. This might indicate inheritance of the disease or of some special tendency to the disease, but on the other hand it might be due to similarity of environment, or again, it might be due to contagion. The question of infection or contagion is necessarily closely connected with that of environment.

"In view of the heavy incidence of rheumatism upon children of the hospital class, it might be expected that the incidence of rheumatism would be in direct relation to the degree of poverty. This, however, was found not to be the case, indeed if the hospital patients were divided into three classes according to the degree of poverty, the poorest of these three, the very poor and destitute, show a lower incidence of rheumatism than the other two. It would seem, therefore, that if poverty is a factor in the incidence of rheumatism, it must be on account of some condition or conditions associated therewith which are not equally present in all degrees of poverty.

"Inquiry was made into the home conditions as regards sleep and play, and general management, maternal care, exposure to cold and damp, clothing, sleeping accommodations, cleanliness, distance from school, also the occupation and the health of the parents and their income. Housing was investigated with reference especially to locality and dampness, the number of rooms per family, the ventilation and sanitary accommodation, and vermin and dust were also considered.

"A comparison between the rheumatic and non-rheumatic children seemed to show that in the rheumatic families the maternal care was less good, and therewith exposure to cold and wet was more common and clothing less satisfactory. There was no evidence of any special frequency of verminous conditions in the rheumatic families, a point of some interest in view of the possibility of conveyance of infection by such means. Dampness, which has been credited with playing a

large part in the causation of rheumatism, was only slightly more frequent in the houses of rheumatic families than of the others.

"Overcrowding of rooms did not seem to be commoner amongst the rheumatic families than amongst the controls, nor was there any appreciable difference between the two as regards sanitation in London, but in Glasgow the proportion of rheumatic families living in houses with bad sanitation was appreciably larger. Neither ventilation nor lighting appeared to be any worse in the homes of the rheumatic families than in those of the controls.

"It may be that an investigation of the conditions under which the rare occurrence of rheumatism takes place in children of the well-to-do whose environment is less similar, would throw into more prominence any common factor of causation. The possibility of direct contagion from person to person is difficult to exclude. If it occurred, it would go far to explain the greater frequency of rheumatism among the children of the poorer classes than amongst those of the well-to-do, for the former live under the conditions of much closer contact.

"In the homes of the hospital children, it was found that commonly two children and often three or more sleep in one bed, and a still larger number of persons slept in one room, and that sometimes of small size. Enquiry showed that in many instances a rheumatic child shared a room or a bed with a sister or brother, or a rheumatic parent slept in the same room as the children. It could not be proved that infection had occurred in this way; on the other hand, individual instances were noted in which it seemed least possible that the disease had spread in this way from one child to another.

"The question is not an easy one to determine, for even in the chronic stage which many of the children had reached, there was no apparent spread to other children by contact; it is still possible that in the earlier and more acute phase there may be a time in which spread by contagion is possible; moreover, contagion if it occurs may be indirect. It is conceivable that a room or a house may be infected by some occupant, particularly by one who was there in the acute stage of the disease.

"Clothes also might be a source of infection, and it was noted that there was less individual proprietorship of clothing amongst the children seen at the hospitals than is usual amongst the well-to-do. The time relationship of the onset of rheumatism in two or more members of the same family was in individual instances suggestive of the possibility of contagion, direct or indirect.

"On the whole while it cannot be said that there is, in the facts collected, any conclusive evidence of contagion, many of them are consistent with this view. Of the many points investigated in the course of this enquiry none has given any decisive indication as to the means which should be adopted for the prevention of this distressing and disastrous disease. Probably more light might be thrown upon the question of the spread of rheumatism, whether by contagion or otherwise, if it were made a notifiable disease.

"This would at any rate secure information as to its prevalence and distribution, matters of importance if institutional treatment upon a wide scale is desirable for children with rheumatism. The difficulty with notification would be considerable, for the diagnosis of the more characteristic manifestations of rheumatism, heart disease, and chorea and even acute articular infection is by no means free from possibilities of error, and in its less pronounced forms rheumatism may be very difficult of diagnosis.

"In the meantime it appears that, although it is difficult to point to this or that fault of environment as responsible for rheumatism, nevertheless, by raising the standard of environment, improving the home conditions so that they approximate to the well-ordered conditions found in such institutions as those in which rheumatism was found to be much less common, and probably by reducing crowding, so that the possibility of contagion may be diminished, that we may hope to reduce the frequency of rheumatism in children."

Bacteriology.—For many years research has been directed toward the isolation of a bacterial cause of rheumatic fever. The generally accepted view of the disease is that it is bacterial in origin, but so far no one microorganism has been proved to be responsible. Many facts, as we have already indicated, seem to bear out the view that rheumatic fever is an infection, and many investigators have isolated microorganisms which they feel to be the specific exciting cause. Poynton and Payne, Swift, Small, Birkhaug and others have cultured streptococci from the blood, valves, joints, pericardial fluid and pharynx of cases and some claim to have produced in animals myocardial lesions very similar to the Aschoff bodies found in the rheumatic heart. It is not necessary here to go into the details of the numerous bacteriological findings. Without doubt, this most important problem will eventually be solved, just as it has been in many other diseases. One salient point stands out: The organism isolated has usually been a non-hemolytic streptococcus; both green-forming and indifferent types

have been recovered. A striking feature has been the variability in cultural and immunological characteristics of the streptococci.

In an attempt to reconcile these findings, Swift and others have advanced a theory which seems reasonable and satisfactory to explain the pathogenesis of rheumatic fever. They suggest that an individual who has an attack of the disease is in a state of allergy to streptococci. One person may be allergic to one strain of streptococcus, while another is allergic to a different strain. This state of hypersensitiveness results, they suggest, from repeated low-grade infections or from the persistence of foci of infection in the body. They point out the well-known fact that, although most individuals are at some time infected with tuberculosis, relatively few ever show clinical signs of the disease. Many individuals who show hypersensitiveness to streptococci never develop rheumatic fever, which is somewhat analogous to the fact that many people who have positive tuberculin reactions never have clinical tuberculosis. Several workers have shown that rheumatic patients give very strong skin reactions to streptococci or their products. Swift and his coworkers, and others, have found that most of their patients with rheumatic fever react strongly to filtrates of both indifferent and green-producing streptococci and to nucleoproteins from all types of streptococci. The individuals with subcutaneous nodules or long-standing heart disease give the strongest reactions. Patients with other diseases also give similar reactions so that this type of skin reaction cannot be considered diagnostic of rheumatic fever. The important fact is that a large proportion of patients with the disease do react. With this conception of rheumatic fever as a result of an allergic condition of the body tissue, it is possible to understand why streptococci, which are so common in the nose and pharynx, do not produce rheumatic fever in all individuals. Only under suitable conditions, that is, if an individual is hypersensitive to streptococci and these streptococci or their products are disseminated to the tissues, is the characteristic picture of rheumatic fever produced. Among these conditions one should consider tonsillitis, sinusitis, or other respiratory infection as probably increasing the reactivity of the patient to some streptococcal product. If, however, the individual possesses a state of immunity without hypersensitiveness, the features of the disease do not appear.

The problem is an extremely complicated one. The allergic nature of tissue reaction to tubercle bacilli is said to be well established, and there are striking similarities between the pathological changes in tu-

berculosis and rheumatic fever. The fact that there are so many strains of streptococci which can be separated culturally and immunologically, compared to the relative simplicity of the tubercle bacillus, makes the problem of reconciliation of the results of many researches a very difficult one. In the light of our present knowledge the view that rheumatic fever is caused by a strain of streptococcus to which a given individual is hypersensitive, seems the most satisfactory explanation of conflicting findings.

CHAPTER III

PATHOLOGY OF RHEUMATIC FEVER

The pathological changes which occur in the heart in rheumatic fever are those which have been most thoroughly studied, and about which most interest has centered. The reason for this is partly that the most profound physiological changes are those which occur as a result of the damaged heart and partly because death does not usually occur while the joints or the brain are acutely affected. As Lessegue has said: "Rheumatism licks the joints and bites the heart."

JOINTS

The pain, redness, swelling and heat in the acute arthritis of rheumatic fever denote an acute inflammation of some sort. At times the swelling is such as to suggest involvement of the joint itself. This is true in some cases, and occasionally fluid collects in the synovial cavity. The inflammatory reaction, however, involves chiefly the peri-articular tissues, and is not a destructive process. The tissues surrounding the joint are infiltrated with a serous fluid, which may become hemorrhagic. There may be ecchymoses in the synovial membrane, with formation of a thin layer of new vascular tissue. Sometimes groups of large cells analogous to Aschoff bodies of the myocardium are found in the neighboring connective tissue. Swift found focal necrosis of the joint-capsule, thrombosis of the smaller arteries, and endothelial and perivascular reaction comparable to the changes in the heart and subcutaneous nodules. A distinction is made between the exudative and the proliferative reactions, two types of response which the body may make to the virus of rheumatic fever.

BRAIN

Since chorea is seldom a fatal condition, the pathology of the disease is not definitely established. Examination of the brain shows in some cases a diffuse encephalitis or meningo-encephalitis. This recalls the fact that choreiform movements are sometimes seen during or following an attack of epidemic encephalitis. The lesions are chiefly around the smaller blood-vessels. Often the lesions are found in the

region of the corpus striatum where injury is supposed to cause choreiform movements. Recently the idea has been advanced that choreiform movements are produced by an afferent disorder of regulation attributable to lesions in the cerebellomesencephalothalamocortical path. The lesions of chorea may include this area.

Hyperemia is the commonest gross finding in chorea. Microscopically, the lesions consist of engorgement of the blood-vessels, thromboses in small arteries and veins and occasionally proliferation and fatty infiltration of the endothelium. There are areas of softening, following the occlusion of vessels, and serous exudation and small round-cell infiltration about the smaller vessels. Changes in the glia and nerve-cells are also found. In some cases vascular and perivascular changes have been noted in the pia-arachnoid. Aschoff bodies have never been found, but this might be explained by the histological structure of the brain. Certainly, the vascular and perivascular changes resemble somewhat those found in rheumatic fever.

SUBCUTANEOUS NODULES

The subcutaneous nodule is pathognomonic of rheumatic infection. On gross examination it is a gray, translucent, highly vascular body,



FIG. 1.—SECTION FROM SUBCUTANEOUS RHEUMATIC NODULE. LOW POWER.
(K. Merritt, *Am. J. Dis. Child.*, Chicago, 1928.)

1 millimeter to 2 centimeters in diameter. The cut surface may show yellowish opacities. There is disagreement among pathologists as to the tissue origin of these nodules, whether they arise from blood-vessels or from fibrous tissue. Some, in fact most, consider the nodules analogous to the Aschoff bodies found in the heart, although

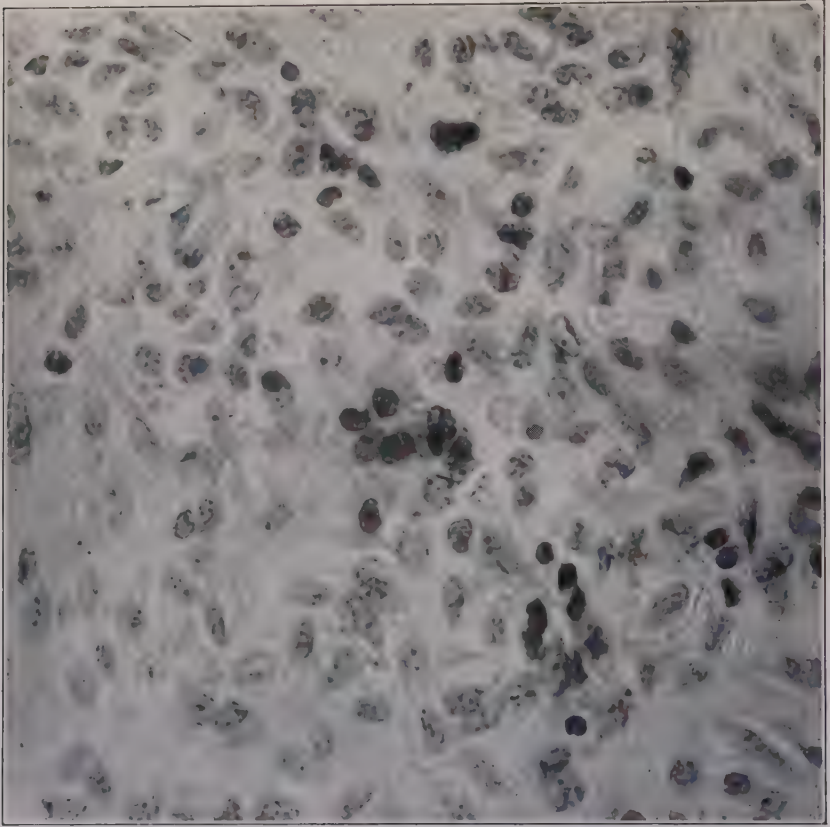


FIG. 2.—SECTION FROM SUBCUTANEOUS RHEUMATIC NODULE. HIGH POWER.

Note multinucleated giant-cell in center of field. (K. Merritt, *Am. J. Dis. Child.*, Chicago, 1928.)

others state that the histology of the nodules does not bear out the analogy. The larger nodules are made up of masses of tiny nodules. According to MacCallum's description, the center of the nodule consists of necrotic tissue, with a small amount of fibrin scattered through (Fig. 1). Some parts show a line of demarcation between the necrotic and the living tissue, where there is a palisade-like arrangement of large cells with irregular cell-bodies and deeply staining nuclei, or several nu-

clei in each cell. The cytoplasm is not granular. Outside this layer there is a "highly vascular mass of branching and anastomosing fibroblast-like cells." There are a few leukocytes in this mass, often with eosinophil granulations and more mononuclear wandering cells. These cells, the nature of which is not certain, have a distinct branching cell-body which takes a purplish stain. The nucleus is large and vesicular, usually distinct, and contains many chromatin particles. Some have several nuclei with nucleoli (Fig. 2). Mitotic figures are often seen in these cells. They are paler than the endothelial cells and seem to form a distinct tissue. They are distinctly and finely branched and attached to each other by these branches. In MacCallum's own words, "there are masses of tissue predominantly formed as a sort of fresh vascular granulation tissue of new fibroblasts concentrated about clumps of necrotic material in the immediate neighborhood of which there is a mantle of peculiar form made up of large, deeply stained, branching cells, which seem slightly different from the fibroblasts and rouse the question as to their possible closer relation to such phagocytic, mononuclear wandering cells or clasmotocytes as are so generally concerned when necrotic tissue is present." Subcutaneous rheumatic nodules may last a few weeks or persist for months. Their disappearance is probably due to absorption.

HEART

Myocardium.—The heart of an individual dying from rheumatic heart disease is always enlarged. The thickness of the ventricular walls is greatly increased from hypertrophy and dilatation, as well as the size of the cavities. Usually the left ventricle is much larger than the right. The papillary muscles are also thickened and lengthened. The actual weight of the heart is increased sometimes to twice that of the average for the age of the patient. The auricles may be thickened, but not to the extent of the ventricles, although the proportionate increase in the size of the left auricle in the case of long-standing mitral stenosis is as remarkable as the huge left ventricle in a case of disease of the aortic valve.

On gross examination the changes in the ventricle walls do not appear to be striking, except for the increase in size, and there is little evidence of inflammatory change (Fig. 3). The muscle in chronic valvular disease is dark, and the veins large and congested. The sub-miliary nodule, or Aschoff body (Fig. 4), is considered the characteristic lesion of the rheumatic heart. These nodules are found chiefly

in the walls of the left ventricle and in the interventricular septum. They occur also in the walls of the right ventricle, and are numerous in the wall and lining of the left auricle. They may be deep in the muscle tissue or near the surface. MacCallum states that in one case

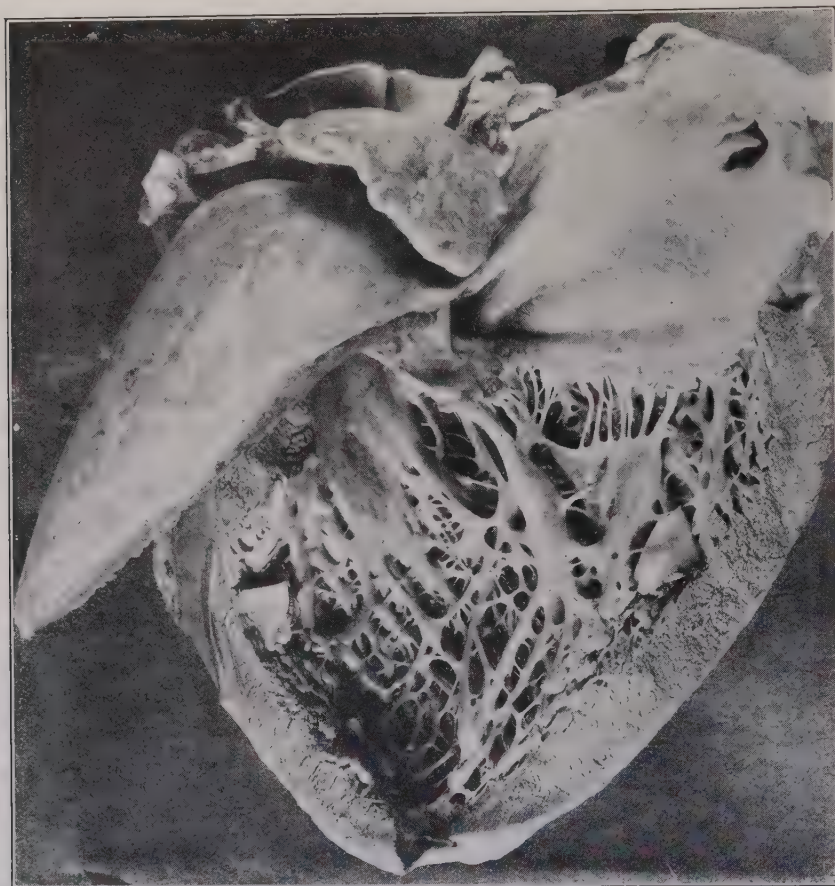


FIG. 3.—HEART OF GIRL (L. W.), AGED 12 YEARS, DYING DURING FIRST ATTACK OF RHEUMATIC ENDOCARDITIS.

Valves were pink and edematous. Vegetations on edges of mitral valve. Dilatation and hypertrophy of ventricles. (Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

he saw numerous pearly white foci scattered especially near the auriculoventricular ring, which on section proved to be Aschoff bodies. These bodies can, therefore, sometimes be seen by the naked eye. They occur usually near arterioles.

The center of the Aschoff body is composed of necrotic material which is probably fibrinous. Surrounding this are numerous large

cells of varying shapes with deeply staining cytoplasm and many nuclei. They are probably not derived from muscle-cells, but from the vascular endothelium, and perhaps sometimes represent new capillary formation. Fibroblasts, plasma-cells and lymphocytes are present. The reaction is an inflammatory and proliferative one, although in the



FIG. 4.—SECTION THROUGH MYOCARDIUM OF SPECIMEN SHOWN IN FIGURE 3.
Six Aschoff bodies in this one field. (Courtesy of C. de la Chapelle.)

early stages this is masked by the many lymphocytes and plasma-cells. Later, when these disappear, the outline of the nodule is definite, with the giant-cells surrounding the central necrotic mass. As the nodule is absorbed and the acute phase dies down it is replaced by fibrous tissue. There may be areas of fibroblastic proliferation aside from the nodules, and the leukocytosis may extend in patches through

the myocardium. The leukocytosis is the result either of passive hyperemia, or of the obstruction which occurs in the blood-vessels.

The blood-vessels show changes which vary from swelling of the endothelial cells of the capillaries and arterioles to a proliferation of these cells, sufficient to obliterate the lumen of the vessel. Sometimes even larger arteries become occluded by a thrombosis. The muscle-cells show some cloudy swelling, but this is not marked. Fatty degeneration occurs both in the acute and the more long-standing inactive cases.

Besides the scar tissue left by the submiliary nodules, there is another type of fibrosis found in the walls of the auricle, which is most marked in long-standing cases of mitral stenosis and auricular fibrillation. It is found in lesser degree in children. Red blood-cells and leukocytes become extravasated between the muscle planes, particularly in the heart which has been rapidly overwhelmed. The arteries and arterioles in the muscle walls become hypertrophied and the veins so distended that the blood-cells escape into the surrounding tissues.

There is a marked proliferation of the subintimal and intimal layers of the arteries and arterioles. The vessels affected are those found near the central fibrous body near the roots of the mitral, tricuspid and aortic valves, and in the proximal half of the valve flaps. The vessels to the auriculoventricular node and bundle are especially apt to be affected.

Endocardium.—*Ventricular Endocarditis.*—Both the mural endocardium and the endocardium of the valves are involved in rheumatism of the heart. Von Glahn has described areas in the left ventricle, which appeared grossly as yellow flecks. Microscopically these flecks were dense collections of nuclei of polymorphonuclear and lymphoid cells, large mononuclear cells and other nuclei which could not be identified. These collections of cells occupied the loose fibro-elastic tissue of the endocardium and projected up on the surface. In some places the endothelium was intact, while in others it had been destroyed. The cells which compose this reaction are the same as those concerned in the changes in the auricular endocardium, but their arrangement is somewhat different. Besides this diffuse reaction there are lesions in the endocardium of the left ventricle which have the same structure and arrangement as Aschoff nodules.

Auricular Endocarditis.—MacCallum, in 1924, drew attention to the fact that the endocardium of the left auricle frequently is the site of characteristic changes in rheumatism (Fig. 5). He felt that

while these lesions were distinctly rheumatic, the Aschoff cells and bodies remained the characteristic lesions. Von Glahn, as the result of his studies, believes that these changes in the auricular endocardium are as distinctive and characteristic a rheumatic phenomenon as



FIG. 5.—ENDOCARDITIS.

Patch of mural endocarditis in left auricle (darker gray patch 1 inch in diameter at right of illustration). Deformed and sclerosed valves with fresh vegetations on edge. (Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

the Aschoff bodies. Although he found these lesions in nine of thirty-five cases of rheumatic valvular endocarditis, he has seen only one case where the right auricle was involved.

On gross examination of an auricle presenting these changes, ridges are seen in the endocardium which may be crowded closely

together or be rather widely separated. They form a plaque of 3 centimeters or more in width. The ridges run roughly perpendicular to the valve ring. Similar ridges can be traced across the auricular surface of the valve to the vegetations near the edge of the cusp.

The color of these plaques is grayish, with a yellowish tinge in



FIG. 6.—THROMBUS FORMATION IN LEFT AURICLE. SCLEROTIC ENDOCARDIUM.

From heart of man, aged 42 years, with auricular fibrillation. (Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

the acute stages. When the process is extensive, there are yellowish ridged plaques over the greater surface of the auricle. Von Glahn states that in one case practically the entire surface was a single slightly wrinkled plaque. On section the endocardium is considerably thickened, and yellowish-gray or gray in color. Sometimes only the superficial part of the endocardium appears to show a change, but histological examination shows that even the apparently normal tissue

below the plaque is much changed. In other cases all layers of endocardium are seen to be involved.

In the older lesions the ridges become flattened and less distinct, but are recognizable as pale gray thickenings sometimes containing a layer of calcium. Section of such areas shows a dense gray homogeneous endocardium, with superficial deposits of calcium. Probably the plaques of calcium which occur in the auricular endocardium in chronic mitral disease indicate these acute rheumatic lesions (Fig. 6).

Histologically, an acute lesion shows edema of the lining layer of the auricle, with collection of various types of cells, including mononuclear cells, polymorphonuclear, and some eosinophils. There is some fibrin formation between the cells. The most striking feature, according to von Glahn, is the arrangement of the large cells which form a palisade along a band of hyaline material, the nuclei of the cells being perpendicular to the band. MacCallum feels that the most distinctive feature is the presence of numerous Aschoff bodies which are forced into rows by the arrangement of the elastic tissue lamellæ, giving the bodies a banded appearance.

In some sections there are accumulations of cells with indistinct outlines and distorted nuclei. These cells do not correspond morphologically with Aschoff cells and are probably degenerated forms of the large wandering mononuclear cells. With these cells are found many polymorphonuclear cells, a few plasma- and small round cells and a few eosinophils.

The tremendous dilatation of the auricular walls which sometimes occurs in long-standing mitral disease has already been mentioned. This dilatation is difficult to explain on purely mechanical grounds, and possibly has its basis in the weakening following acute rheumatic infection of the walls.

Valvular Endocarditis.—An acutely inflamed valve has a pink color, is swollen, and has a fibrinous deposit. This fibrin is formed in a narrow line near the edge of the cusps. In the auriculoventricular valves, this fibrin is on the auricular surface; in the semilunar valves it is on the ventricular surface. There may also be deposits of fibrin in other parts of the valve, and on the chordæ tendineæ.

The vegetations which occur on the valves in rheumatic endocarditis are typical (Figs. 7 and 8). They appear as tiny cauliflower or wart-like excrescences of about the same size, 1 to 2 millimeters in diameter, and are grayish, pinkish-gray, or grayish-white in color. They are situated along the line of closure of the valve. Occasionally they are

somewhat larger and extend for a few millimeters along the chordæ tendineæ. In the early stage the vegetations are dull and opaque, but later become glistening. In the mitral valve the entire line of closure is usually covered with vegetations, but in the aortic and tricuspid valves, only part of the valves may be affected. The vegetations are



FIG. 7.—SCLEROSSED MITRAL AND TRICUSPID VALVES, WITH FRESH VEGETATIONS ON BOTH.

The aortic valve, which is not shown in this view, had the same condition. There is fibrinous pericarditis with a localized patch of adherent pericardium at apex. The myocardium was studded with Aschoff bodies. From a young woman, aged 17 years, who died in third attack of rheumatic fever. (Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

tough and, although they may be rubbed off, they adhere with considerable tenacity to their point of attachment. This explains, according to Libman, why the vegetations do not cause embolism, which, in rheumatic carditis, is usually due to thrombosis of the left auricle (see Fig. 6). Evidence is accumulating, which shows that the changes on the surface of the valves are preceded by a proliferative interstitial

reaction in the subendocardial tissue. Coombs describes the reaction as follows:

"Proliferation occurs in the fixed connective tissues, resulting in a free formation of fibroblastic cells, and blood-vessels are rapidly produced, being presumably projected into the valve from these vessels which normally penetrate but a short distance into its basal part. Around the newly formed vessels fibroblasts are arranged in concentric circles. Their endothelium attacked by toxin, swells up, proliferates, and is shed. Thrombosis follows, and a submiliary nodule may be produced. The center is occupied by a fibrinous area: around this,

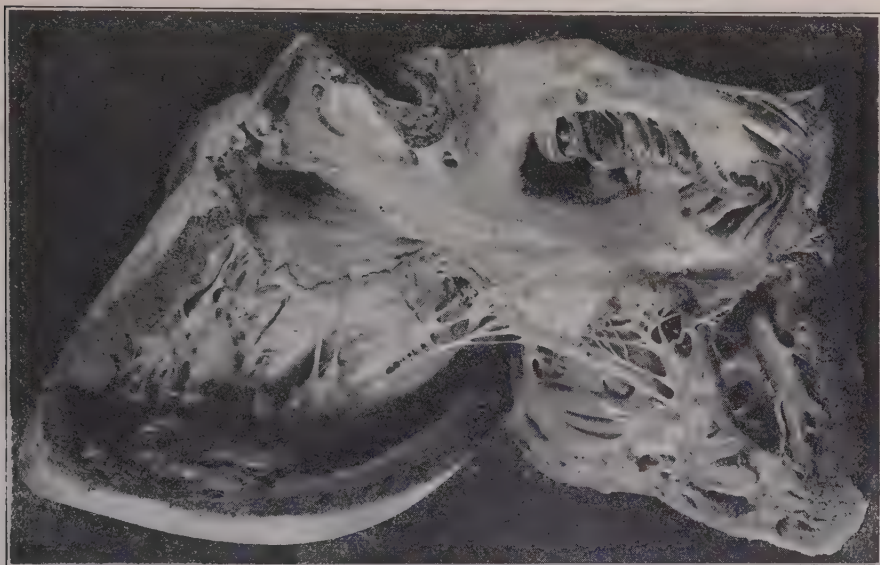


FIG. 8.—TRICUSPID VALVE: OLD AND FRESH INFECTION.

Note line of vegetations on edges of valve. (Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

forming a border which separates it from surrounding parts, is a more or less regular circle of large cells, many of them multinucleate. This again is often enclosed by a belt of fibroblasts, interspersed with a few leukocytes. The exudative phenomena play a subordinate part as a rule. Leukocytosis is mild and diffuse. In most instances it is mononuclear in type, though in the severest degrees of carditis, polymorphonuclear may equal or even predominate over lymphocytic and plasma-cells" (Figs. 9 and 10).

It is suggested that the virus of rheumatic fever is carried to the valves through the intrinsic vessels and that this is anatomically possible has been shown by the works of Swift and of Kugel and Gross.

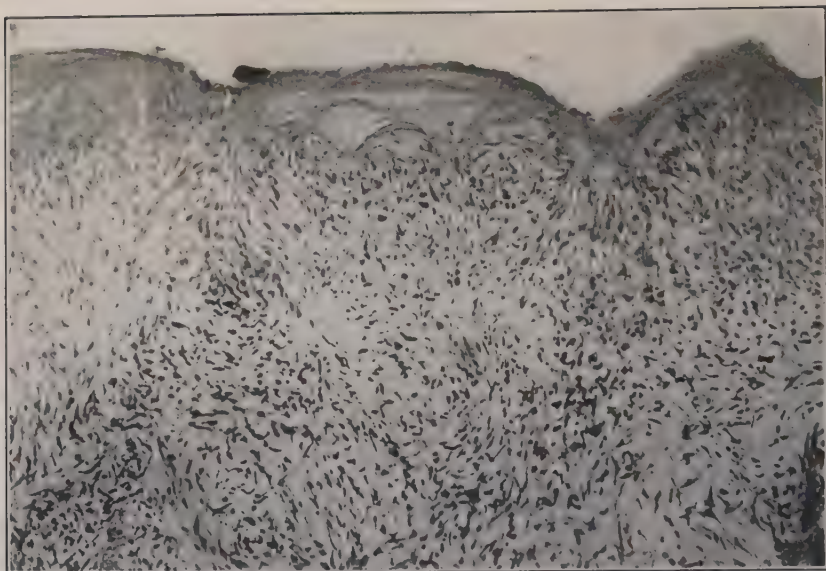


FIG. 9.—SECTION THROUGH EDGE OF ACUTELY INFLAMED MITRAL VALVE.
Thrombotic elements at edge of valve. Diffuse cellular reaction in body of valve.
(Courtesy of C. de la Chapelle.)

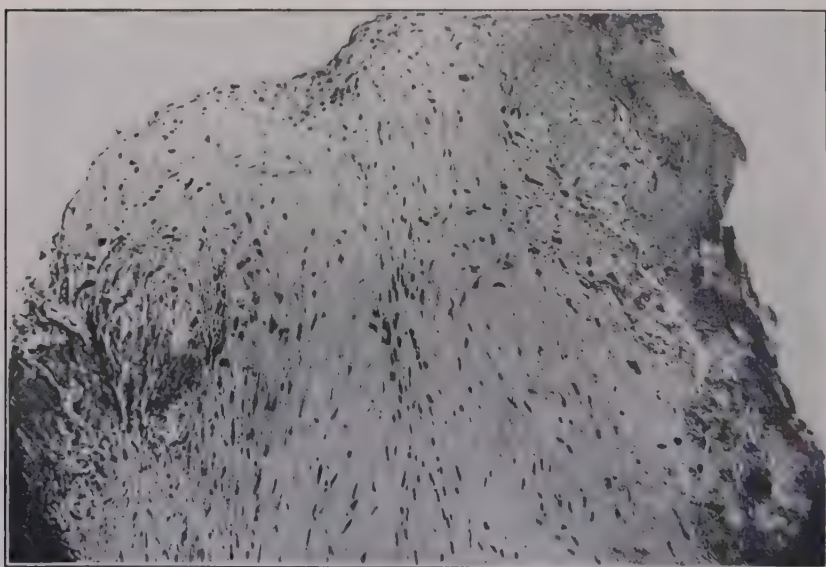


FIG. 10.—SECTION THROUGH SCLEROSED MITRAL VALVE WITH FRESH VEGETATIONS.
Young woman, aged 19 years, who died of rheumatic fever, bronchopneumonia
and some signs of congestive failure. (Courtesy of C. de la Chapelle.)

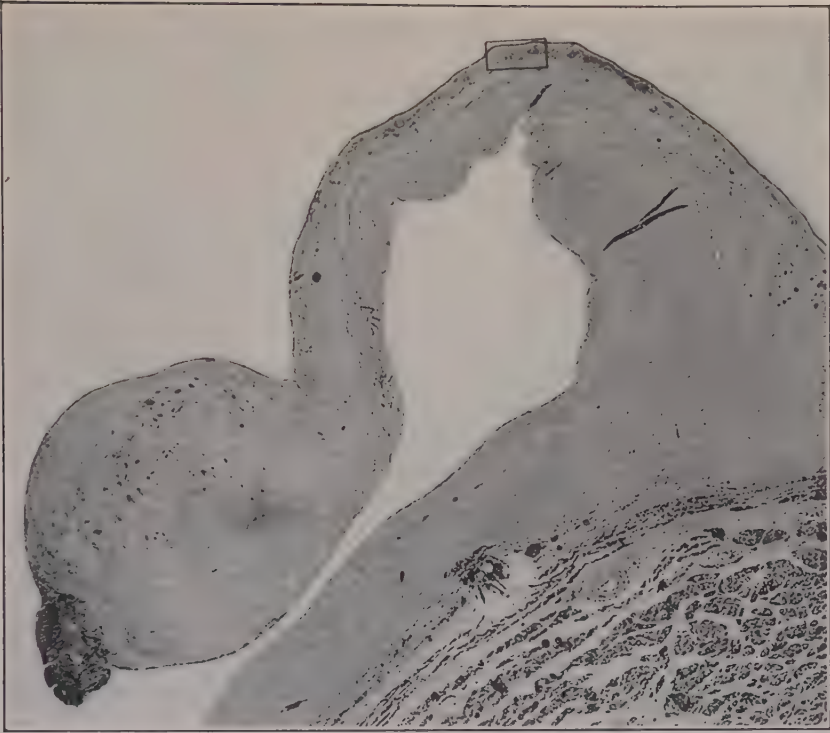


FIG. 11.—CHRONIC RHEUMATIC VALVULITIS, AORTIC VALVE.

Low power; showing thickening, vascularization, and a thrombotic verruca at the free margin. Small area shown high power in Figure 12. (Courtesy of Homer F. Swift.)

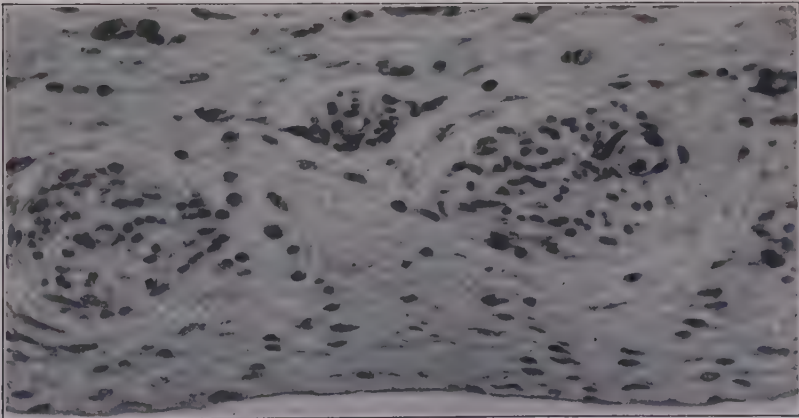


FIG. 12.—PERIVASCULAR CELLULAR REACTION AROUND VESSELS PROBABLY FORMED AT TIME OF PREVIOUS INFLAMMATION.

Compare Figure 11. (Homer F. Swift, *J. Exper. M.*, New York, 1924.)

In understanding the permanent effects of rheumatic disease of the heart valves it is important to realize that the inflammation is not only an endocarditis, but a valvulitis extending far beyond the area of the attachment of the vegetations. Healing appears to begin at an early stage: blood-vessels and fibroblasts invade the verrucæ,

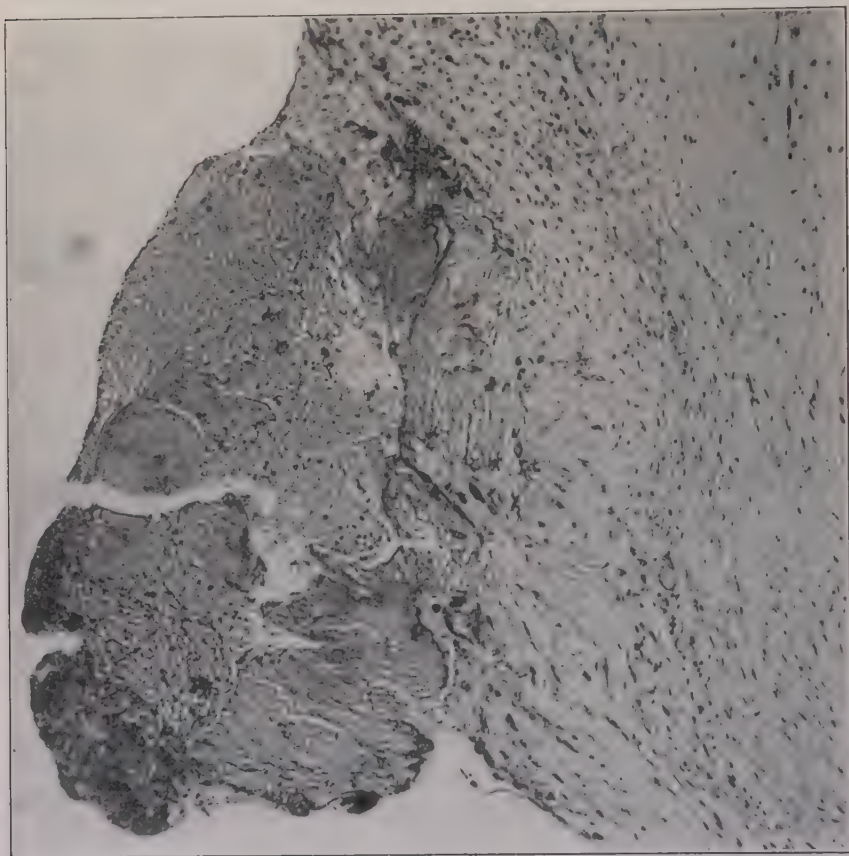


FIG. 13.—THROMBOTIC VERRUCA MADE UP OF FIBRIN, PLATELETS AND SOME RED BLOOD-CELLS DEPOSITED ON AN AREA OF GRANULATION TISSUE.

(Homer F. Swift, *J. Exper. M.*, New York, 1924.)

and cicatrization finally occurs (Figs. 11, 12, 13). In the mitral valve the greatest degree of permanent scarring results. The cusps are thickened, particularly at the free edge; they become shortened and drawn up towards the auricles, eventually becoming stiff and immovable as a result of the fibrosis. The cicatricial changes which shorten the chordæ tendineæ at the same time and in the same manner as those in the valve segments, prevent accurate closure of the valves.

The endocardial folds which pass from cusp to cusp become thickened and shortened, so that the mitral channel is invaded from all sides. This means that in some advanced cases the aperture through which the blood is forced is a rigid slit between the stiff valve cusps, instead of a through channel which varies in size in the different



FIG. 14.—ADULT'S HEART (AURICULAR SIDE), SHOWING MITRAL AND TRICUSPID STENOSIS.

(Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

phases of the cardiac cycle (Fig. 14). When looked at from the auricular side, this narrow slit has a buttonhole appearance. From the ventricular side the cusps are seen to project down on to the ventricular cavity to give the funnel-shaped appearance. However, fibrosis of the mitral valve does not always cause narrowing of the channel because the walls of the ventricle become weakened by the lesions in the musculature, so that the ventricle and the auriculoventricular rings

become stretched. In spite of the cicatrization, the mitral channel is thus often held open, making the opening larger than normal and allowing regurgitation. The tricuspid valve shows the same type of lesion, except that marked fibrosis, with stiffening, shortening and deformity, rarely develops.

When the aortic valve is inflamed, usually all three cusps are involved, although the degree of inflammation varies. The cusps are thickened, especially near the free edges, which are curled and relatively rigid. In very severe cases the stiffened cusp may project rigidly into the aortic channel. Permanent injury to the pulmonary valves is very rare. Microscopically, the valves which become scarred show dense fibrous tissue formation, containing a few blood-vessels chiefly near the edge. The endothelium returns to normal. Lime deposits are found deep in the cusps. Coombs emphasizes the fact that both active and old changes are frequently found in the same valve at the same time. A valve that has been once injured is more likely to be injured again. He states that "it is more usual than not to find evidence of recent inflammation visible to the naked eye in valves already deformed and sclerosed." In the first decade, 87.5 per cent of all rheumatic valvular lesions show active changes; in the second, 75 per cent; in the third, 43.4 per cent; and, taking all ages together, 51.6 per cent.

The changes described, namely, acute inflammation followed by cicatrization, may occur in any or all of the valves of the heart. The mitral valve is almost always involved. Table IV shows the incidence of involvement of the different valves. It will be seen that the tricuspid valve is found at autopsy to be damaged more frequently than is generally realized. Libman and Sacks found fresh vegetations

TABLE IV
COMPARATIVE INCIDENCE OF VALVE INJURY IN RHEUMATIC ENDOCARDITIS

VALVES	COOMBS: 97 Cases		VON GLAHN: 109 Cases 48 (44 Per Cent) Males 61 (56 Per Cent) Females			
	Number	Per Cent	Acute	Chronic	Total	Per Cent
Tricuspid	35	36	19	26	45	41.3
Pulmonic	2	2	3	1	4	3.3
Mitral	97	100	42	57	99	90.8
Aortic	57	58.7	20	43	63	57.7
Myocardium	—	—	46	28	74	67.8
Myocardium only	—	—	8	—	8	7.3
Pericardium	—	—	7	34	41	37.5

on this valve in 66.6 per cent of their cases (a series of eighteen cases of endocarditis with Aschoff bodies in the myocardium).

In the early stages of rheumatic infection of the heart the most commonly made diagnosis is mitral insufficiency. The pathological changes in the mitral valve itself have very little, if anything, to do in producing the inability of the valve leaflets to close. The myocardium weakened by infection allows the rings to stretch, and this or shortened chordæ tendineæ produce the insufficiency of the valve.

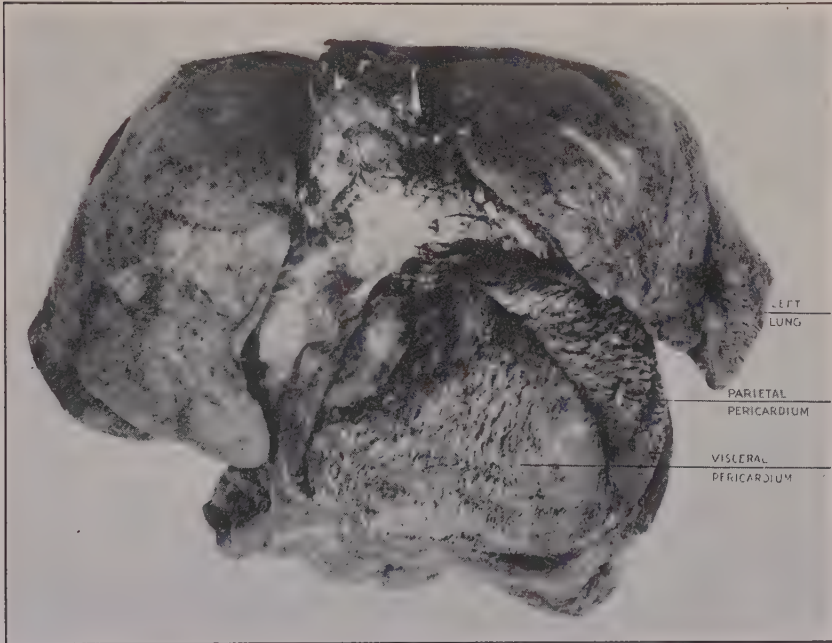


FIG. 15.—ACUTE FIBRINOUS PERICARDITIS, WITH PATCH OF DENSELY ADHERENT PERICARDIUM AT APEX.

Heart of S. C., aged 7 years. Note shaggy appearance of fibrin on both layers of pericardium, great enlargement of heart and compression of lungs.

Pericardium.—Fibrinous pericarditis is one of the most typical lesions of rheumatic infection. According to Coombs 53 per cent of cases of all ages show inflammation of the pericardium at autopsy, although clinically it may not be demonstrable. He states that all children dying in the first decade showed pericardial lesions, and 83.3 per cent in the second decade. There may be a collection of fluid, sometimes turbid, never purulent, and almost never occurring in quantities sufficient to indicate paracentesis. The average amount of fluid found by Coombs was 4 to 5 ounces. Usually pericarditis is accompanied by

myocarditis and endocarditis. Sometimes the inflammation occurs in small areas, but commonly the whole pericardium is involved. In early



FIG. 16.—ADHERENT PERICARDIUM.

Heart of boy, aged 14 years, who died of heart-failure. Pericardial layers are densely adherent to each other throughout. Mitral and aortic stenosis (not shown in this photograph). Microscopic section revealed no sign of active infection. (Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

and mild cases a thin layer of fibrin exudes on the pericardial surface. When there is a large amount of fibrin, the action of the heart beats it into a series of ridges running transversely or obliquely or in a network which gives the heart a characteristic shaggy appearance (Fig. 15).

The serous membranes beneath the fibrin are hyperemic and sometimes ecchymotic.

Friable adhesions develop between the visceral and parietal layers of the pericardium, which later become organized, dense and firm (Fig. 16). In very severe cases the parietal pericardium may become adherent to the pleura, diaphragm, mediastinum and chest wall. Microscopically, the exudate is composed of fibrin, leukocytes, platelets and erythrocytes. The subpericardial tissue is edematous, becomes invaded with capillaries and fibroblasts, and shows a diffuse cellular reaction, particularly around the smaller vessels. The vessels show often endothelial swelling and proliferation, sometimes with thrombosis, and may become thickened as a result of the deposition of the hyalin-like material or fibroids in their walls.

Aschoff bodies are nearly always present and are often large. They are most frequently and characteristically found during organization of the pericarditis. In general, the features of rheumatic pericarditis are the same as the myocarditis, when allowance is made for the anatomical structure of the pericardium, and the way in which serous membrane reacts to inflammation.

BLOOD-VESSELS

Aorta.—The gross appearance of the aorta is normal, but microscopically Pappenheimer and von Glahn found Aschoff cells or nodules in the adventitia, and dense scars, often acellular, in the region of the nutrient vessels. In the acute stage they found striking changes in the adventitia, and outer two-thirds of the tunica media. The endothelial cells of the vasa vasorum were swollen and about these vessels were collections of small round cells, plasma-cells, and polymorphonuclear leukocytes. In parts of the adventitia, the infiltration of cells was diffuse and intense. No definite Aschoff bodies were found. The changes in the tunica media were essentially the same as found in the adventitia, except that the distribution appeared to be determined by the course of the nutrient vessels. The walls of the nutrient arterioles were thickened, due partly to swelling and partly to the heaping-up of the endothelium, possibly also to the cellular infiltration and edema of the walls. Near the nutrient vessels were collections of cells, lymphocytes, polymorphonuclears and Aschoff bodies. Pappenheimer and von Glahn feel that the findings in this case support the view that scars found in the tunica media are preceded earlier by focal perivascular lesions.

Coronary Arteries.—The changes in the coronary arteries are not constant, and according to MacCallum are common only in severe cases, although other workers consider the changes more frequent. They may occur as a result of the presence of contiguous nodules which develop in, or extend into, the tunica media. Sometimes the lesions occur in vessels which have no relation to Aschoff bodies. Endothelial proliferation occurs in the coronary arterioles in mild cases, sometimes with infiltration by round cells into the intima, with or without narrowing of the lumen of the vessels. In more advanced cases the endarteritis is more pronounced, and then there may be thrombosis and involvement of the tunica media.

Occasionally there is thrombosis without evident changes in the walls of the vessel or there may be marked changes in the vessel without thrombosis. In very severe cases there is actual destruction of the elements of the vessel wall, to the extent even of tiny aneurysmal pouching. The small- and medium-sized vessels are more often affected than the larger ones. In a few cases small infarcts appear to have been the result of occlusion of numerous small vessels. Marked fibrous intimal thickening in the small arteries of old rheumatic hearts is not uncommon. Coombs found that the artery to the auriculoventricular node often was almost completely obliterated.

Other Arteries.—When we realize that distinctive changes in the aorta are found in rheumatic infection, it is not surprising to find lesions in the smaller peripheral arteries also. Von Glahn and Pappenheimer examined in detail forty-seven consecutive cases dying of rheumatic carditis and, in ten of these, they found changes in the peripheral vessels which they believe to be specific and characteristic. These vascular changes were found in the lungs, aortic valve, kidney, perirenal and periadrenal adipose tissue, appendix epiploica of the sigmoid colon, ovary, testis, pancreas, and in a small polyp of the cecum. Only isolated vessels were affected in most of these areas, although in two cases practically every small branch of the pulmonary arteries was involved. The changes in the kidney were also widespread. They did not study the subcutaneous fat, joints and skeletal muscle. They found exudation of fibrin in and about the vessels, destructive changes in the cellular elements of the vessel walls, distinctive cellular reaction in the adjacent tissue and absence of thrombosis. No Aschoff bodies were found. The acute changes were followed by organization, with or without the formation of new collateral channels in the intima and sometimes in the muscular layer.

Veins.—Venous thrombosis is not a common complication of rheumatic fever, but it does occur. The most usual sites are the veins of the neck and upper extremities. A recent case at Bellevue Hospital was a girl, aged eight, who had an extremely severe rheumatic infection, with subcutaneous nodules, fever, polyarthritis, and congestive failure. The venous thrombosis occurred in the right upper extremity, and preceded death by only a few weeks. In most of the reported cases definite clinical evidence of active infection was lacking. In the absence of histological study, it is impossible to say whether specific lesions occur in veins.

LUNGS AND PLEURA

A common complication of rheumatic carditis is fibrinous pleurisy, which occurs most frequently on the left side, occasionally on both sides. The incidence of this complication is between 5 and 10 per cent in most series of cases. There may be a serous effusion which rarely calls for paracentesis. Organization of the exudate which follows the acute inflammation leads to the formation of adhesions between the parietal and visceral pleura. In the presence of severe pericarditis these adhesions may be very extensive and involve all the surrounding tissues.

Pneumonia may complicate rheumatic fever, and the question of a specific rheumatic pneumonia has been discussed. Rabinowitz concluded that a special type of pneumopathy which is not a true pneumonia, occurs during rheumatic fever, usually in cases of pancarditis, but sometimes independent of arthritis or carditis. The lesions at autopsy, he stated, are congestion, edema and atelectasis, not true pneumonic consolidation. In this connection the findings of von Glahn and Pappenheimer of vascular lesions of the pulmonary vessels may be illuminating.

Paul found Aschoff bodies in the adventitia of the pulmonary artery as well as of the aorta, and changes such as those described by von Glahn and Pappenheimer in the arterioles of the lung in 20 to 40 per cent of active cases. Recently, Eiman and Gouley examined the lungs of two cases. They found that both lungs were enlarged and bulky, did not collapse and sections sank in water. The cut surface was very smooth, fairly dry and edematous, and oozy in the less involved areas. There was no crepitation. The lesion resembled atelectasis, but there was no gross evidence of collapse. They summarized their findings as follows:

"The histological study of these lungs showed an acute interstitial inflammatory process, apparently hematogenous in origin, with hyperemia, edema and a characteristic cellular exudate, perivascular, consisting of large endothelial cells, giant-cells, plasmacytes, lymphocytes and a few polymorphonuclears, the distribution of this exudate and its general appearance being identical with the cardiac lesions in rheumatism, and occurring in such a case."

These findings are very important in considering the question as to whether there exists a true rheumatic pneumopathy.

Formerly the explanation of pulmonary signs in the lung during an attack of carditis was given as infarction. At autopsy, it is rare to find pulmonary infarcts in rheumatic fever, although they are not uncommon in subacute bacterial endocarditis. When infarction does occur it is in long-standing cases of heart disease where thrombi have formed in the auricles. An embolus comes from such a thrombus, not from the vegetations in the heart.

KIDNEYS

It is common to find urinary changes in rheumatic carditis, but whether there is a true nephritis has not yet been proved. Chronic diffuse glomerulonephritis is rarely found at autopsy, so that if acute nephritis occurs it is necessary to assume that the cases recover. Nephritis is common, however, in subacute bacterial endocarditis. The vascular lesions in the kidneys have already been mentioned.

SUMMARY

From the foregoing brief descriptions of the pathological changes found in the body in rheumatic fever, it will be seen that these changes are by no means confined to the heart. They are found in many tissues of the body and further studies may show other organs to be specifically involved. It is important to remember that specific rheumatic changes are not confined to the heart and that the proper conception of rheumatic fever is not that of a disease consisting primarily of an arthritis with heart disease or chorea as complications, but of a disease which may attack any of several tissue simultaneously or successively. It is true that involvement of the heart produces the most profound interference with function not only of the heart, but of the whole organism. For that reason, from a clinical standpoint, consideration of the heart frequently overshadows other factors. As

far as treatment of the child goes, this may make no difference, but an accurate appraisal of pulmonary signs, for instance, is more likely if the examiner realizes that there probably exists a rheumatic pneumopathy with typical pathological changes. The physiological changes which follow as a result of permanent damage to the tissues will be discussed in another chapter.

CHAPTER IV

PATHOLOGICAL PHYSIOLOGY

Knowledge of the changes in physiology of the heart action is much more difficult to determine than of the anatomical changes which occur as the result of damage to the heart by disease. Since the development of instruments of precision such as the electrocardiograph, our knowledge of disordered cardiac function has greatly increased. Before discussing the morbid physiology the normal physiology of the heart will be considered briefly.

Normal Physiology.—The heart is divided into halves, each side consisting of an auricle and a ventricle, which normally have no direct connection with each other. The right auricle receives blood from the venæ cavæ, sends it to the right ventricle which through its contractions forces it out to the pulmonary artery. The blood is collected by the pulmonary veins, taken to the left auricle which sends it to the left ventricle which forces it into the aorta. The auricles contract simultaneously, for about a tenth of a second, followed by a pause of about the same length. This is followed by contraction of the ventricles, which lasts about three-tenths of a second. The period of relaxation of the muscle walls, or diastole, lasts about four-tenths of a second. These figures are based on observation of adult hearts. The heart rate of children is more rapid than adults, varying somewhat by age and sex, so that the time of both systolic contraction and diastolic relaxation must be shorter. Studies of the time which it takes the impulse to pass from the sino-auricular node into the ventricles in children (measured by the electrocardiogram) seem to show that this period is briefer than in adults. During diastole, the blood flows from veins into auricles and from auricles into ventricles. As ventricular contraction begins there is a rapid rise in pressure during which the auriculoventricular valves close. The pressure in the ventricles continues to rise. As soon as it becomes greater than the pressure in the aorta, the aortic valves open, allowing the blood to enter the aorta. The pressure caused by contraction of the auricles is transmitted along the veins. The functions of the heart valves are,

in general, to prevent reflux of blood, and to avoid interference with the flow of blood in the heart.

The first heart-sound is probably a combination of the contractions of the wall of the ventricles and of the stretching and closing of the auriculoventricular valves. The second heart-sound is caused by the sudden closing and stretching of the aortic valve and occurs at the end of systole as the ventricle stops contracting. The pulmonary and aortic valves close simultaneously, although sometimes the aortic closes first. This may explain the occasional "reduplicated second sound" heard at the base.

Mechanism of Heartbeat.—The initiation of the heartbeat normally takes place in the sino-auricular node, which lies at the junction of the superior vena cava and the right auricle. The excitation wave spreads in all directions from this node simultaneously through the muscle-fibers of the auricles, and thus reaches the auriculoventricular node. This node lies at the edge of the auricular tissue at the posterior and right border of the septum. From the auriculoventricular node the wave passes to the auriculoventricular bundle. The bundle lies ensheathed in a canal and extends to the interventricular septum. Near the tricuspid valve the bundle divides, into right and left branches. The left branch remains ensheathed on the upper border of the septum, and when it reaches the left ventricle beneath the right anterior and posterior cusps of the aortic valves, it enters the subendocardial space. From here it spreads downward in the left side of the septum, sending two main branches to the papillary muscle of the mitral valve. The right division soon becomes subendocardial, proceeds downward on the right side of the septum and goes to the papillary muscles of the right ventricle. Here it breaks up into arborizations. The arborizations on both sides are directly continuous with the network of Purkinje fibers which lies beneath the endocardium and which extends through the greater part of both ventricles. These fibers connect directly with the muscle-fibers and are specialized tissue designed to carry the excitation wave rapidly through the ventricles.

The impulse which causes the heart to contract, then, begins at the sino-auricular node, spreads through the muscle-fibers of the auricles, reaches the auriculoventricular node, proceeds to the auriculoventricular bundle, thence to the right and left branches and through the arborization of these branches connects with the Purkinje fibers in the ventricles. The time which is consumed in the passage of the impulse from auricle to ventricle is represented in the electrocardiogram by

the P-R interval which normally ranges from 0.12 to 0.2 second. The importance of knowledge of this interval in rheumatic heart disease will be brought out later.

PATHOLOGICAL PHYSIOLOGY

Mitral Valves.—*Insufficiency* of the mitral valve, that is, the inability of the valve to prevent reflux of blood into the left auricle during ventricular systole, is the commonest type of disordered heart function in the early decades of life. That this is not due principally to actual changes of the valve is shown by the fact that at autopsy there is found only slight deformity of the valve in recent cases. The insufficiency is due to the *myocardial factor* which allows stretching of the ring and thus regurgitation of blood. Shortening of the chordæ tendineæ may also prevent closure of the valves. There are definitely many children who have rheumatic heart disease with a mitral stenosis, and an insufficiency which is due to dilatation of the mitral ring. If in such a case the leaflets of the valves were not shortened by the sclerosing process, they might be able to compensate for the stretching of the ring. Probably the stiffness of the valve cusps has some effect on the loudness of the murmur in mitral insufficiency.

That *stenosis* of the mitral valve constitutes an obstruction to the flow of blood from auricle to ventricle is obvious. There are degrees of stenosis, which may be arbitrarily described as: (1) The stage seen in childhood and early adolescence where the heart is enlarged. There is some thickening of the cusps, and usually fresh vegetations. (2) The condition found in late adolescence and youth where the heart is still enlarged, the cusps are definitely thickened, but because of the stretching of the ring there is little narrowing of the channel. (3) The stage found in adult life when the cusps are rigid, thick and deformed and the mitral orifice is small. At this stage the heart is not much enlarged because the increase in work is not thrown on the ventricle.

The accentuated first sound which is always present in early rheumatic carditis is due to the increased size of the ventricle walls. In mitral stenosis this accentuated sound also has a valvular quality. This is caused mainly by the sharp systolic contraction resulting from imperfect filling of the ventricle during diastole. Another factor is the tensing of the auriculoventricular cusps and chordæ tendineæ, which is more sudden than normal because of their shortened and thickened state.

The “reduplication” of the second sound at the apex or inside it,

which is so often heard in the early rheumatic heart, is frequently a third heart-sound. It is, however, louder and more definite in cardiac rheumatism than the normal third sound. It must be differentiated from the short mid-diastolic murmur which is often heard in real mitral stenosis and which is due to obstruction to the flow of blood through the valve in diastole. This latter may be somewhat less valvular than the third heart-sound, and with an increased heart rate often becomes presystolic.

Distention of the auricle occurs as the result of mitral-valve insufficiency, which, as we have seen, is myocardial rather than valvular in origin. The tone of the walls of the auricle is diminished by this distention, and partly also by the invasion of the auricular wall itself by the rheumatic infection. The resulting physical signs are dulness up and to the left in dilatation of the left auricle and to the right with obliteration of the cardiohepatic angle in dilatation of the right auricle. Auricular failure, as evidenced by total arrhythmia (auricular fibrillation), is not common among children, although it does occur. It will be discussed fully in the chapter on electrocardiography.

Accentuation of the pulmonic second sound is due to increased tension in the pulmonary artery which results from auricular stasis and also to the pushing away of the lung, which leaves the artery in contact with the chest wall. As the auricle stretches the pressure of the pulmonary veins increases. The capillaries become distended, and the pressure in the pulmonary artery and its branches increases. This explains the symptoms of dyspnea and cyanosis in long-standing mitral heart disease. The increase in intrapulmonary pressure may cause insufficiency of the pulmonary artery, but this is rare.

With the stretching of the auricle, the formation of clots is possible, particularly in the auricular appendage, and it is this clotting which is the source of the emboli which cause occlusion of cerebral or other arteries. Thrombus formation in the auricle is more apt to occur in adults than in children since the mitral stenosis is of longer duration. Right auricle thrombosis may give pulmonary emboli.

Tricuspid Valve.—Great widening of the tricuspid ring is frequently found at autopsy and is the factor, rather than disease of the valve, which causes insufficiency of the valve, just as in the case of mitral insufficiency. This insufficiency allows regurgitation of the blood into the right auricle with resulting stasis in the systemic vessels, cyanosis, engorgement, pulsation and tenderness of the liver and other

organs. Stenosis of the tricuspid valve rarely is a factor in disturbance of cardiac function.

Aortic Valve.—In contrast to the myocardial factor which allows insufficiency of the mitral and tricuspid valves, the *insufficiency* of the aortic valve is produced by disease of the valve cusps themselves. Although the aortic valve appears to be less susceptible than the mitral to invasion by the organism which causes rheumatic fever, less injury will cause incompetence. The result of failure of the aortic valve to close adequately during diastole is overfilling of the left ventricle, since the volume of blood which enters the left ventricle from the left auricle is augmented by the blood which flows back from the aorta. This causes dilation of the left ventricle, followed by hypertrophy. The greater the regurgitation, the greater the hypertrophy, since the left ventricle must have greater force to send out the blood into the aorta.

The effect of aortic regurgitation on the peripheral circulation is to produce pressure in the capillaries, where normally there is no pressure and no pulse. This leads to capillary pulsation. There is a great fall in blood-pressure in the arteries due to reflux of the blood during diastole or an increased "pulse pressure," the difference between the systolic and diastolic pressures. Finally, the tissues fail to receive sufficient nutrition and symptoms such as headache, malnutrition and muscular weakness appear. As Coombs says, "Except for the final phase of breakdown when the systolic pressure begins to fall, the pulse pressure affords a more direct measure of the degree of reflux than does any other means of inquiry; for the relatively low diastolic pressure is a direct measure of the extent to which the aortic valve leaks and lets the bottom fall out of the diastolic arterial column, thus lessening the tension within the aorta and its branches during the period of diastole." In some children with great anemia all the physical signs of aortic insufficiency may be present, but at autopsy only a stretched ring and normal valve cusps are found.

Stenosis of the aortic valve occurs but rarely in children. When present, the effect is to hinder the flow of blood from ventricle into aorta, with production of characteristic physical signs. This lesion gives enormous hypertrophy of the left ventricle because it has to work directly against the obstruction. The systolic murmur heard so frequently in connection with the murmur of aortic insufficiency, but without the other signs of stenosis, probably means a certain small amount of stiffening of the cusps so that the flow of blood is obstructed enough to cause a murmur but without much real back pres-

sure (so-called aortic roughening). This perversion of function in such cases is much slighter than the effects from the regurgitation.

Pulmonary Valve.—That there may be regurgitation from the pulmonary valve is recognized, but it is rare as a result of direct injury to the valve. Secondary regurgitation from increased pressure in the pulmonary artery due to mitral stenosis and pericardial adhesion does occur. This throws added burden on to the right ventricle, but when this happens the heart is usually so overwhelmed that the final stage has already been reached. The murmur produced is a diastolic bruit in the pulmonary area.

Pericardium.—When the pericardium is acutely inflamed, the myocardium is at all times also involved. The inflamed pericardium is not so resistant to pressure, but this is of less importance functionally than the weakness of the myocardium. In pericarditis with effusion the amount of fluid in the pericardial sac is seldom great enough to interfere with function. It is the formation of adhesions which really interferes with the working of the heart. The extrapericardial adhesions constitute more of a burden to the heart than the adhesions between the two layers of pericardium. Adhesions between the pericardium and surrounding tissues are evidence of a very severe infection, in which the heart itself is usually involved and is therefore unable to function normally. If, in addition, it has to pull against adhesions, function is very greatly impaired. For a time hypertrophy of the walls compensates for this, but final breakdown is inevitable.

Myocardium.—The chief function of the heart is ventricular systole, by which the blood is forced from the heart out into the general circulation. Anything which interferes with this function necessarily causes changes in the physiology of the heart action. We have already shown that insufficiency of the mitral valve is chiefly due to stretching of the mitral ring rather than to changes in the valve leaflets themselves. In the early stages of rheumatic carditis when the infection is still present in the heart, failure depends on the changes in the muscle walls which affect contraction of the ventricles. Later, when there is stenosis of the mitral valves, an added burden is put on the muscle, but the effect is indirect rather than direct. Large deposits of fibrin on the pericardium also add some burden to the ventricle, but it is later, when the fibrin organizes with the formation of adhesions between the pericardial layers and other neighboring tissues, that the pericarditis constitutes a real contribution to failure. Loss of tone of the ventricular walls leads to dilatation, but there may be recovery

from this if sufficient contractility is maintained. Loss of the contractile power of the ventricles leads eventually to death.

Evidence of disturbance of the metabolism of the muscle-cells is found in the signs of dilatation of the heart, tachycardia, dyspnea and other signs of heart-failure. Signs of congestive failure may also occur in the presence of active infection of the heart muscle. Premature contractions do not occur much more often in acute carditis than in the apparently normal heart, but if persistent, may perhaps indicate some focus of irritation. Sinus arrhythmia is so common in normal children that it has no significance in those with carditis.

Disturbance of the conduction of the excitation wave from auricle to ventricle is evidence of inflammation somewhere in or near the conduction system. This may be in the sino-auricular node, the auriculoventricular bundle or anywhere along the conduction path and it may be due to inflammation of the artery to the auriculoventricular bundle. This disturbance in conduction is termed "heart-block," and may be of varying degrees. The first degree, prolongation of conduction time, is called first-stage block. It is not demonstrable clinically, but is very clearly shown in the electrocardiogram. If electrocardiograms are taken frequently during attacks of rheumatic fever, a lengthening of the P-R interval will be demonstrated in a high percentage of cases. This finding definitely indicates acute inflammation in the heart, even in the absence of clinical evidence, and is therefore very valuable information to have (see Chap. XI).

The second stage of block is evident clinically and consists of dropped beats, *i.e.*, the failure of some of the impulses to reach the ventricle. The commonest is 2:1 block. In third degree block none of the impulses reach the ventricle, which therefore establishes its own independent rhythm (about thirty beats to the minute), while the auricle goes on contracting at its usual rate.

The first and second stages of block probably represent some focal inflammation which subsides, since the rhythm returns to normal after a few days or weeks. It may be the only definite evidence of continued myocardial involvement. A boy, in Bellevue for a rather mild attack of acute rheumatic polyarthrititis, showed second degree block during the early stage of his attack. For some weeks following he had occasional slight rises in temperature, and a continued moderate leukocytosis, with an absence of definite clinical signs of infection. His general condition improved steadily. The dropped beats persisted for only a few days, but the electrocardiograms showed that first degree block

lasted for a period of several weeks. Such a case demonstrates the importance of prolonged bed care of these children even when they appear to have made a complete recovery.

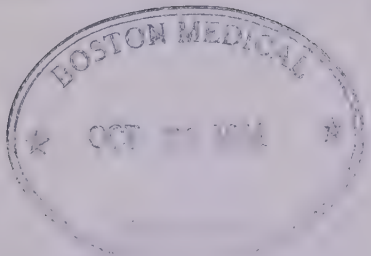
Failure of the auricle, as evidenced by auricular fibrillation, occurs in the later stages of heart disease, and is seldom seen in children, except those approaching adolescence. The mechanism of fibrillation is similar to that of flutter, and is the result of "circus movement," a change in the course of the excitation wave through the auricle. It shows clinically in total irregularity of the heart rhythm, with stimuli being produced so rapidly in the auricle that only parts are transmitted to the ventricle by the conduction system. This disturbance of the normal physiology is caused by the changes in the walls of the auricle. The excitation waves, instead of being able to proceed normally through the auricular muscle, are blocked, and begin to go around and around in a circle. The stimuli which are thrown off pass irregularly through the auricular walls, causing them to fibrillate. Quinidin acts by depressing the excitability of the auricle so that the circus movement is stopped. Digitalis is effective by decreasing the conductivity of the auriculoventricular system so that the ventricle contracts at a rate compatible with life, but it does not directly stop the circus movement as quinidin does. If the ventricular wall has not already been too badly damaged, cessation of rapid and irregular stimuli will allow the heart to recover from the attack of auricular failure.

Sometimes marked sinus arrhythmia plus frequent premature contractions will produce an irregularity which simulates auricular fibrillation, but if any great doubt exists, an electrocardiogram will solve the problem.

The mechanical factors which affect the already damaged ventricle wall are pericardial adhesions, sclerosis of the aortic valves and stenosis of the mitral valve. Pericardial adhesions affect both right and left ventricles, aortic sclerosis results in overdistention and overworking of the left ventricle, while mitral stenosis raises the intrapulmonary tension so that the right ventricle becomes overworked. Venous stasis in the heart itself results from chronic distention of the right heart which causes chronic distention of the coronary sinus. In adults there is also the normal deterioration of the myocardium and blood-vessels to add to the burden of the chronic rheumatic heart.

Failure of the ventricle is less dramatic than failure of the auricle, but it is upon the condition of the ventricle that the efficiency of the heart depends. Edema of the lower part of the body is the most char-

acteristic sign of failure of the left ventricle but it occurs only when some extra burden, such as a fresh infection, or pericardial adhesions, is put on an already deteriorated myocardium. Extreme cyanosis, enlargement of the liver and dyspnea are signs of failure of the right ventricle, but they occur only during very severe attacks of carditis in children, and in the final stages of rheumatic heart disease in adults.



CHAPTER V

RHEUMATIC FEVER

There are certain acute illnesses which are considered to be of rheumatic origin, that is, each one is thought of as a different manifestation of infection by the same organism which may at the same time or at some later date attack the heart. Although they are spoken of as different conditions, it is permissible to group them all under the term rheumatic fever. Although they will each be described here as more or less distinct clinical entities, in actual practice it is not possible so to separate them. The diagnosis of one of the minor conditions may be made certain by the presence of one of the other conditions. For instance, a boy recently seen in the clinic was found to be pale and looking generally less well than on previous recent visits. His temperature was normal and he had no special complaints. He had had a mitral insufficiency and stenosis for some months. Careful examination showed, in addition to the signs of organic heart disease, an erythematous rash on the left arm and lumbar region of the back. Also, over the right patella there was a moderate-sized subcutaneous rheumatic nodule, and another small one over the left olecranon. Without the definite heart lesion the rheumatic nature of the erythema would have been questionable and the diagnosis of rheumatic nodules less certain. Taking the whole picture, however, we could be sure that the rheumatic infection was active in this child.

It was found that of 112 children with chorea on the wards of Bellevue Hospital during the years 1926 and 1927, twenty-nine, or 24 per cent, gave a history of joint pains at the onset or just prior to the beginning of the chorea, or had such pains during the course of the chorea. The presence of chorea made the rheumatic origin of even the mild pains certain. In going over the charts of 399 outpatient-department cases of heart disease, it was found that 107, or 26.8 per cent, had had both chorea and acute rheumatic fever in the past (Table V).

Similarly rheumatic carditis must certainly occur in some cases with involvement of no other organs as far as can be determined clinically. Out of 427 cases of heart disease in outpatients at Bellevue, 78, or

TABLE V
OCCURRENCE OF RHEUMATIC INFECTIONS, NOT INCLUDING CARDITIS, WITHOUT
OTHER MANIFESTATIONS

Infection	Cases	Per Cent
Chorea	74	18.5
Polyarthrititis	95	23.8
Chorea and polyarthrititis	107	26.8
Growing pains	34	8.5
Joint pains	89	22.3
Total	399	

78 cases (18 per cent) out of 427 (both sexes) of organic heart disease gave no history of polyarthrititis, chorea, growing or joint pains.

20.9 per cent of 253 girls.

14.4 per cent of 174 boys.

18.2 per cent, gave no history whatever of chorea, acute polyarthrititis, or growing pains. On the other hand, recent investigations have shown that cases of acute rheumatic fever show electrocardiographic changes which indicated invasion of the heart muscle. The tracings were taken at frequent intervals, and changes were discovered which would otherwise not have been found. Quite often, a soft systolic blow may be heard over the apex of the heart during an acute attack of rheumatic fever, which disappears with convalescence. The same occurs frequently during an attack of chorea. We are probably justified in considering that most cases of acute rheumatic polyarthrititis have also some degree of heart involvement which may subside entirely for the time being. It is therefore evident that, although each of the rheumatic manifestations may be described individually, they are not separated clinically.

The diseases which are included as rheumatic infections are:

1. Acute rheumatic polyarthrititis
2. Chorea
3. Carditis
4. Recurrent sore-throat, or tonsillitis
5. Subcutaneous nodules
6. Growing pains
7. Skin lesions

ACUTE RHEUMATIC POLYARTHRITIS

Symptoms and Signs.—Acute rheumatic polyarthrititis as we see it in childhood is a different picture from that presented by the adult, where acutely inflamed and very painful joints dominate the picture.

The *involvement of the joints* in children may cause swelling, redness and exquisite tenderness, but more commonly this does not occur. The joint pain may be severe even when there is no heat or swelling, but is usually of short duration. The joints most commonly involved are the knees, ankles, elbows, wrists and shoulders, either singly, together, or successively. Any joint may be involved.

There is almost invariably *inflammation of the pharynx*, including the tonsils, if they have not been previously removed. Sometimes the child complains of a sore-throat, but more frequently he does not. The acute inflammation of the throat usually subsides within a few days.

Fever is always present at the onset of the attack, and may go up to 102° F., rarely much higher than this. The fever usually subsides in a few days and remains around 99° to 100° F. for a time. After this it may be normal for a week or longer, but very frequently, almost as a rule, there are brief flare-ups of the temperature to 100° to 102° F. at irregular intervals, and for no apparent clinical cause. That is, there may be no complaints such as an acutely inflamed joint concomitant with the rise in temperature. It is important to watch for these rises in temperature, as they probably signify activity of a latent infection.

Other *minor symptoms* associated with illness due to infection occur, such as headache, poor appetite, and general malaise. Very often, for a few days or longer, before the acute onset of the attack the mother will notice that the child becomes unduly fatigued, may be short of breath or simply does not act quite as lively as usual.

Precordial pain is a frequent symptom during an attack of rheumatic fever. Swift and Hitchcock studied ninety-six such cases in an attempt to determine the relationship of this symptom to the development of heart disease. A wide variation in the severity of the pain was found; in some cases the precordial symptoms were so mild in comparison to the joint symptoms that only great care on the part of the observers determined their presence. When the infection is severe with extensive involvement of the heart the pain resembles angina pectoris, probably indicating involvement of the aortic valve and even of the aorta itself. The precordial pain may occur in the absence of physical exertion. Children in the clinic not infrequently complain of precordial pain; in such cases it seems to be related to excessive physical activity. Sometimes it is the first symptom which indicates impending decompensation. Whenever it occurs it is an important

symptom. Swift and Hitchcock separated their cases into groups according to the severity of the associated conditions. They found that 37 per cent of the ninety-six patients with polyarthritis had precordial pain or tenderness; 80 per cent of fifty-eight patients with carditis or nodules had precordial symptoms: "It is evident at once that the incidence of these symptoms increases hand in hand with the liability to cardiac involvement as indicated by the severity of the infection."

Two-thirds of the patients in this series of Swift and Hitchcock presented other evidence of cardiac involvement, and most of the rest showed evidence of cardiac involvement during some period of the infection. A greater number with precordial pain had permanent valvular disease than those who were free from such pain. The great frequency with which the precordial pain and cardiac involvement occurred together, indicates that cardiac pain in a patient with rheumatic fever should be interpreted as an indication for treatment designed to save the heart.

Another symptom which frequently occurs early in an attack of rheumatic fever is *abdominal pain*. It is not unusual to get the history that the child first complained of abdominal pain and was given a cathartic, had fever, and then a few days later developed pains in the joints. The significance of this symptom in relation to rheumatic fever is not clear, and no statistical studies appear to have been made. Epigastric pain in children with already developed heart disease is likewise often encountered.

The *duration* of an attack of rheumatic fever is variable. The monophasic type lasts about two weeks. The child comes with fever and acute joints. The symptoms subside under treatment, the temperature goes to normal and remains there. The polyphasic type is more common. The onset is similar to the monophasic type, but the temperature, after a few days' subsidence, flares up again. With the secondary rises in temperature there may be severe or fleeting joint symptoms, or the fever may indicate development of pancarditis or a flare-up of myocarditis. Very frequently, however, the fever recurs without any other clinical signs of continued infection. The fever may be no more than one or two degrees, but it is extremely important to watch for these rises, since, in the absence of any other cause, they indicate a still active process in the body.

Laboratory procedures are of importance in rheumatic fever, particularly the white blood-cell count. At the beginning of an attack, the white cells are always increased, although rarely over 15,000. Weekly

white blood-cell counts are extremely valuable in determining the child's actual condition. If the leukocyte count remains slightly above normal and there is an occasional slight rise in temperature, the child needs continued bed care. *Vital capacity* (or "lung capacity" as determined by the spirometer) gives additional evidence as to the child's condition. It is often found that preceding the attack of rheumatic fever, perhaps several weeks before any symptoms appear, the vital capacity begins to diminish. Such a case should be watched at close intervals to catch the first definite evidence of chorea, polyarthritis or carditis. Moderate *secondary anemia* is almost the rule. The rheumatic child is pale, has dark circles under the eyes, and gives evidence of undue fatigue in the slumping posture. The urine is usually highly concentrated, acid and may show albumin, with white cells, and even casts and red cells.

The *weight curve* is an important guide in handling these children. When they are on the mend the weight goes up, while the infection is still active the weight goes down or is stationary. At the onset they are almost always malnourished, and the picture which the rheumatic child presents is very similar to that of the tuberculous child.

The *pulse rate* in children is not an infallible guide. Premature contractions frequently occur, and sinus irregularities are common even in the absence of disease. Heart-block, partial or complete, may result from invasion of the auriculoventricular node or the fibers of the conduction system by the rheumatic infection. A slight rise in temperature may send the pulse rate up, out of proportion to the amount of fever. In general, the pulse rate may be taken as one factor in sizing up the evidence for or against allowing the child out of bed after an attack of rheumatic polyarthritis.

The point which deserves the most emphasis is the question of determining whether the rheumatic infection is active or not. If the infection is active, the child needs bed care. If the child is symptom-free, has a normal leukocyte count, has had a normal temperature for at least two weeks, a fairly stable pulse rate, a rising weight curve and increasing vital capacity, it may be considered that recovery has been instituted.

Treatment.—The child with acute rheumatic fever must be put to bed immediately, and be kept absolutely quiet. This is an important part of the attempt to prevent the development of a permanent lesion of the heart. Salicylates are indicated for the relief of pain and for pyrexia. Local applications of methyl salicylate relieve the pain of the acute arthritis. The affected joints should be wrapped in cotton to

avoid the pain which occurs when the joint is touched. The fact that acute rheumatic polyarthritis appears to be less severe in height of temperature, joint involvement and duration of symptoms than formerly may be because of the use of salicylates, which controls both the joint pains and the fever. The true condition may be masked by the continued use of the drug. For this reason it is important to omit the drug for a week or ten days before allowing the patient out of bed. Otherwise a child with a still active infection may be allowed up and about, thus increasing the probabilities of the development of heart disease.

The dose of salicylic acid, usually used as sodium salicylate, aspirin or novaspirin, may be begun with 10 grains every three hours and worked up to as much as 100 grains a day if indicated and if the patient can tolerate it. Sodium bicarbonate should be given with each dose of the salicylate, twice as much soda as of salicylate, to avoid nausea and other toxic symptoms. Some recommend that the initial dose of sodium salicylate be larger. Codein may occasionally be needed if the pains are severe. The daily amount of salicylates may be decreased as the symptoms subside. Toxic symptoms should be avoided if possible. Neocincophen in some cases gives more satisfactory results than sodium salicylate. The dosage is 5 grains, with double the amount of sodium bicarbonate, four or five times daily.

The general condition of the child must be considered, and to this end a liberal high calorie diet is given after the very acute phase is over. Cod-liver oil should be given to the poorly nourished child, and if the appetite is meager, tonics may be prescribed. For the secondary anemia which is present so frequently, an iron tonic is indicated.

Diet and Regimen.—In treating acute rheumatic fever, as in every other acute disease, it is important to lay down a regimen which is to be followed rigidly by patient and nurse. Written directions should always be given the nurse so that there may be no question as to the orders. This should go without saying, but needs mention because it is more honored in the breach than in the observance by most physicians. The diet during the febrile stage should be simple, easily digestible, and given in light meals at frequent intervals. It is best composed of cereals, gruels, cream soups, fruit juices, milk toast, bread, ice cream. One of these given every three hours is enough while the fever is high. After it breaks, milk may be added, later vegetables, cooked fruit, eggs and finally meat. The latter is not given until the temperature is normal, not because meat has anything to do

with rheumatic fever, but because it is not the best food for any febrile case. The diet interval of three (or four) hours best determines the interval of other treatment.

The temperature (rectal) should be taken just before feeding, and any treatment, medication and so forth, should be given right after feeding. This "bunching" of treatments disturbs the child less than if the temperature is taken every four hours, food given every three hours and medicine every two hours. Under such a plan the poor child has no chance to rest.

The local treatment of the nose and throat must be remembered in every case. This is necessary because there is often a local infection in rheumatic fever and also because all febrile cases are apt to accumulate stagnant mucus in the nasopharynx. It does no harm, at least, to instill every three hours into the nostrils the following medication:

Menth $\acute{o}$ l	gr. i-ii
Camphor	gr. i-ii
Eucalyptol	gr. i-ii
Petrolat. q.s.	ad $\frac{3}{4}$ i

This should be done with the head held back on the edge of the bed until it is practically inverted. In this position the oil runs to the top of the nasal cavity, then back to the pharynx. Instilled in the ordinary position, with the child lying flat on his back, the oil merely flows back along the floor of the nose.

When there is definite infection, especially with a purulent discharge, argyrol, 10 to 15 per cent, or other silver salt should be used two to three times a day. It is remarkable how often nothing is done for the nasopharynx in acute illness and how quickly a purulent discharge will clear up at times if properly treated in this simple manner.

The child's bowels must never be forgotten. It is well to give a laxative each night when the patient is in bed, to be followed by an enema in the morning *if no movement takes place by 10 A.M.* This is necessary because the laxative may have been insufficient. No infection can be fought efficiently if the intestinal tract is loaded. Mothers and nurses are apt to be too optimistic in this matter and to hope that perhaps the bowels will move "later in the day." This is unphysiological and results in absorption of toxic products and delayed recovery.

The laxative to which the child is accustomed is the one of choice. Milk of magnesia works well with many, but other children object

bitterly to its taste. Aromatic extract of cascara, senna syrups, phenolphthalein or even the despised patent medicines are justifiable if they work and if the child will take them without a struggle. Palatableness is an important matter. Castor oil and calomel are not necessary except rarely at the onset.

The usual quiet, sun, air and so forth, which are desirable in the sickroom, need only be mentioned. Rheumatic fever cases often perspire profusely and are better off between light blankets or even in a light wool sleeping garment. The wool does not get clammy when wet, as does cotton. This does not imply a large amount of heavy bedding.

If possible, the child who has recently had an attack of rheumatic fever should be sent away for convalescent after-care for a period long enough to insure a state of good nutrition and also to learn good habits of diet and hygiene. When the child goes directly home from the hospital to his old environment he is presumably exposed to the same factors which produced the original infection. This is assuming that the disease is an infection which is transmitted in some as yet unknown way from person to person. One concept of rheumatism is that it is a latent infection which remains in the body and which has acute exacerbations at varying intervals after the first manifestation. It seems a possibility that the recurring attacks may be actual reinfections. Certainly it is true that an individual who once has had an attack of acute rheumatic fever is prone to have recurring attacks. If the child must go directly home from the hospital, it is the responsibility of the social worker to investigate the home conditions and improve them if possible. If the home is dark, ill-ventilated and damp, the family should be urged to move to more hygienic quarters. If the child is in the habit of sleeping with another member of the family, the parent should be advised to provide a separate bed for him, not only for his own sake but for the sake of other children in the family, who might contract the infection from him. Such changes are difficult to make, particularly as the intelligence of the class from which a majority of these patients come is often not high enough to allow them to grasp the desirability of the change. Also, their economic condition frequently makes a move to better quarters impossible. This makes it all the more important to keep the child away from his habitual environment for as long a period as possible.

Whether or not the heart has been evidently involved in the attack of rheumatic fever, the child should be kept under observation in the

cardiac clinic so that the earliest signs of heart disease or of recurring rheumatic infections may be detected. This surveillance should be continued for many years, perhaps even for life, since patients in adult life who think themselves perfectly well may develop signs of rheumatic heart disease, even though the only rheumatic manifestation may have occurred in childhood many years before.

CHOREA

Symptoms and Signs.—The initial symptoms of chorea are almost always the same. The mother notes that the child is “getting nervous,” drops his spoon, spills food, cannot button his clothes. The teacher finds him nervous, fidgety and irritating because he is unable to sit still or do simple tasks. As a result she scolds the child and thus increases the nervous symptoms.

The onset is sometimes sudden, after falls, accidents or frights. This has helped to strengthen the old idea that chorea is primarily a nervous disease, due to nervous causes. The idea that it is purely an infection or the result of an infection was slow to take root.

A typical attack of chorea is unmistakable. The extremities are in constant motion except during sleep. The child grimaces, is unable to control the muscles of the face so that the expression is often so vacant as to suggest imbecility. Intelligible speech may be impossible or absolute aphasia may exist for months. The arms cannot be held extended steadily, and may be dropped immediately after lifting. The hand grip is weak and unsustained and voluntary movements are impossible. Frequently one side is more affected than the other and leads to the complaint of paralysis of one arm.

The attack usually begins gradually with unstable emotions. The child is irritable, cries easily, and perhaps drops objects or spills food. The symptoms gradually increase in severity until the characteristic condition appears. In some instances the attack comes on suddenly in the fully developed form. Chorea is often spoken of as a disease of school children because the incidence is so much greater during this age period.

A very severe case may simulate a hemiplegia. The child lies perfectly quiet in bed until voluntary motion is attempted. On examination, one hand or leg, or both, are found to be practically powerless. The hand grip is so weak as to be almost absent. If the arm is raised from the bed, the child is unable to hold it up. In this form the aimless, wild, constant movements typical of chorea are absent. Occasionally,

the differential diagnosis between chorea and a hemiplegia is difficult. In one girl not quite three years old with a history of lues in the parents, there was an apparent paralysis of the right arm and leg. Her nutrition and general condition were good, and it was thought that she had a hemiplegia. The condition improved slowly under rest. For the two following years, however, she came to the hospital with typical chorea, and now has a definite organic heart lesion. She had a negative Wassermann test and no physical signs of congenital syphilis.

There are no characteristic changes in the reflexes in chorea. The knee-jerks are sometimes markedly exaggerated and sometimes difficult to obtain at all.

The spinal fluid shows no characteristic changes, and there are no signs of a meningitis. The leukocyte count is usually slightly above normal and there is some irregular slight rise in temperature. The urine is normal.

The general condition of the child with chorea has been the subject of comment, and it is generally thought that it is only or chiefly found in thin, poorly nourished individuals. We see it frequently, however, in perfectly well-nourished children, even in those who are overweight.

The significance of an attack of chorea lies in its relation to the development of heart disease. A child seldom dies from an attack of chorea and for that reason the pathology of the disease has not been studied often. Its frequent association with other rheumatic manifestations, that is, carditis, subcutaneous nodules and acute polyarthritides, and the frequency with which heart disease occurs subsequently, give it a distinct importance.

There is a group of cases which give such a definite history of overwork in school, of sudden fright or some other emotional crisis just prior to the development of chorea, that it may well be possible to differentiate two types of chorea, one infectious, caused by the same organism which causes acute rheumatic fever, the other non-infectious. Until the causative organism is positively identified this differentiation will be impossible to prove. The four attacks of chorea in one child were precipitated in each case by a death in the family.

The duration of an attack of chorea varies from two or three weeks up to several months. Sometimes the case becomes chronic, with remissions of only a few days or weeks, over a period of years. Very often the mother gives the history of an attack of chorea occurring at about the same season for a period of several years.

A useful test for chorea is to have the child grasp both the examiner's hands firmly and to have him try to keep perfectly still. He may do so at first, but after a short time the irregular contractions of his fingers will be felt. This may be brought out by asking the child a question which requires an answer. The activity of his speech center may bring out the choreiform movements.

Differential Diagnosis.—Chorea must be differentiated from the ordinary habit spasms of childhood and from other chronic nervous conditions which simulate it.

Habit spasm is frequently seen in nervous, thin children. It differs from chorea in that the same act is repeated again and again. This may be a twitching or wrinkling up of the nose, nodding the head, shrugging the shoulders or almost any movement. But while the movement made may change from time to time, it is generally the same movement repeated over and over in a coördinated manner. A choreic child cannot possibly repeat the same motion twice and all the movements are absolutely incoördinated and purposeless. The child with habit spasm can refrain from his pet trick if he tries hard, but the harder the choreic tries the worse he gets.

The simple tests of grasping the examiner's hands, holding the fingers extended, bring out the chorea after a few seconds, while the habit spasm rarely appears on these tests. The child with habit spasm does not drop his spoon and can button his clothes. It is necessary to bear in mind this differentiation because of the difference in treatment. Nearly every year we see children kept in bed for chorea and their parents greatly alarmed about the possibility of heart disease, when the child has simply a slight habit spasm. The latter may make a child seem very twitchy and a teacher may easily be puzzled, but there is no excuse for a physician confusing the two conditions. We have seen several children who were treated for chorea, who had some central nervous-system condition simulating it.

The postencephalitic choreiform group resembles chorea remarkably, probably because there is an actual lesion in the same region of the brain. But there is usually the history of the encephalitis and the condition is chronic or very prolonged. One boy was admitted to Bellevue Hospital in the spring, at the height of the chorea season. He had typical incoördinate movements, but was able to talk and feed himself. He was treated as a mild chorea, put to bed behind a screen and kept very quiet. All the other choreas in the ward gradually improved and it was a surprise that after several weeks he was no better. A

more careful neurological examination showed that his movements, while typically choreic, could be stopped by his will for almost an indefinite period. In other words, they were not increased by the usual tests, but were arrested. He had no signs of rheumatic fever. A diagnosis of basal ganglion lesion was made, possibly postencephalitis.

Several cases have been seen in whom there were choreiform movements lasting from birth. The movements were suggestive, but not typical. They could be arrested by intention, but returned at once on relaxing. One child is extremely choreic in appearance, yet rides a bicycle, plays ball, writes and is very bright in school. He has had these movements since birth and has probably had a birth hemorrhage in the basal ganglion.

Treatment.—*General Management.*—The first requisite in the treatment of chorea is rest, both physical and mental. The patient should be kept in bed alone in a quiet, well-ventilated room and other children not allowed to play with or disturb him, until convalescence has begun. If the movements are very wild, the sides of the bed should be high to avoid falling out on the floor, and well padded so that the extremities will not be injured. All patients except in mild cases have to be fed for a time. A high calorie, well-balanced diet should be supplied, supplemented by cod-liver oil if necessary. Plenty of water should be given. All emotional excitement must be avoided. The child who is unable to talk and thus communicate his needs or desires, is apt to become very excited and cry wildly. This can be avoided only by careful attention to nursing details, and attempts to guess what the child wants. The diet during the very acute stage of an attack of chorea should be simple and easily digestible, and fluids should be forced, particularly in the severe cases, to avoid acidosis or dehydration. Later a full-balanced diet with cod-liver oil may safely be given. It is very necessary to build up the child's general condition by whatever means are available. Alpine lamp therapy is of distinct value, particularly in the anemic undernourished patients. As in all cases confined to bed especial care to avoid constipation is necessary. A laxative each night and an enema should be given in the morning if needed, as well as a laxative diet.

Drugs.—Many different drugs have been used in chorea, and this fact alone shows the lack of uniform effectiveness of any one drug. Bromids in our experience have been ineffective. Salicylates in large doses are sometimes used, but their value is doubtful. Luminal at times quiets the child. If pushed to toxic doses (fever, rash, etc.),

it may stop the chorea, but it is not without danger. In extremely active cases chloral hydrate may be necessary. Warm baths or cold packs may be used in very severe cases. Small felt that he obtained good results with his serum in cases of chorea.

After-Care.—With patients who are convalescing from chorea, it is important to keep them occupied while they are still in bed. Weaving, basket making, coarse embroidery and similar constructive activities may be given them, which will keep them happy, prevent the development of masturbation, and reëducate the muscles in coördination. Reading may be allowed, but not highly exciting adventure and mystery stories. It is sometimes difficult to decide upon the right time for allowing the convalescent chorea out of bed, even when the temperature and blood-count are normal and there is no evidence of an active carditis. Some choreic movements may persist for a long time. It seems at times as though the continuation of the movements were from habit, and in such cases the child may be tentatively allowed out of bed. If the chorea becomes worse, the patient must be put back in bed, but very often the movements stop with increase in general activity. When the time comes for the child to leave the hospital the question arises as to the advisability of sending him home. Very frequently, if the child returns to his old environment, the chorea will recur in all its severity. This may be a reinfection or an exacerbation of the same infection. In general it is much safer to send the child to a convalescent home for as long a period as possible before letting it go back to his own home, unless the conditions here are definitely known to be good.

The child who has had chorea should be kept under regular supervision, preferably in a cardiac clinic where he will be classified as a potential cardiac, if the heart has not yet become involved. It is important to keep the general health in as good condition as possible in order to prevent further infection. The teeth, sinuses and the pharynx must be kept healthy, overfatigue must be avoided and the nutrition carefully watched. It is obviously desirable to detect any heart lesion as early as possible, and for this reason the child should visit the clinic at least four or five times a year for a careful examination.

CARDITIS

By the term carditis is meant the acute inflammation of the heart itself which occurs from a rheumatic infection. This inflammation may affect the valves, the myocardium of the ventricles and auricles

as well as the endocardium and pericardium. The symptoms may be very mild, and the condition easily escapes recognition unless the patient has acute rheumatic polyarthrititis or chorea at the same time, or the carditis may be the only manifestation of the rheumatic infection, with symptoms of very severe involvement of the heart. It is essential to a true understanding of rheumatic infections to realize that rheumatism of the heart is a definite entity and may be diagnosed even when unaccompanied by other rheumatic phenomena and with no history of rheumatic fever in the past.

Symptoms and Signs.—In a mild case, the only symptoms may be undue fatigue, pallor, loss of weight and appetite, and slight dyspnea on exertion, which is usually noticed by the mother rather than complained of by the child. The true cause of these symptoms is frequently overlooked unless the child is examined by a physician who has this syndrome in mind, and who makes a thorough examination. A large number of cases of heart disease are discovered merely in the course of routine health and school examinations or in dispensaries to which the child is brought for a minor complaint. In perhaps the majority of these there is absolutely no history of any joint symptoms, chorea or other rheumatic disease. A few may give the story of "growing pains" or fugitive, insignificant joint pains.

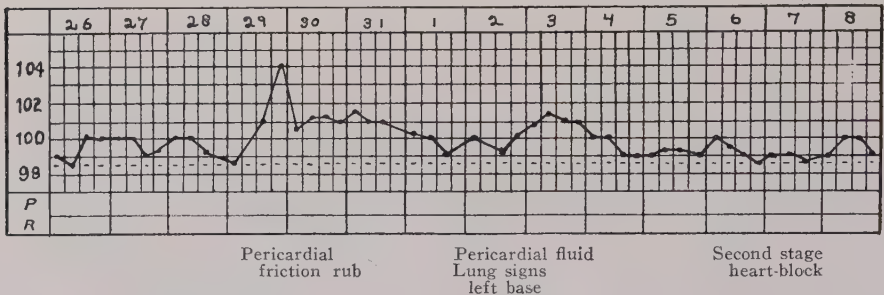
On examination the child will be found to be pale, with dark circles under the eyes, and probably a moderate secondary anemia. The nutrition is usually poor, or at least less good than is normal for that individual, and the posture is almost invariably bad. The heart rate is increased, and there may be marked pulsation noted in the neck. The apex-beat is plainly visible and diffused over several interspaces. This is more noticeable in a thin-chested child than in an adult with a thicker thoracic wall. In the first attack, the heart will not have become hypertrophied, and the apex-beat, though diffuse because of poorly acting muscle, is not forceful. The time at which the heart begins to enlarge is difficult to determine. Coombs believes that the hypertrophy begins as the acute inflammation starts to subside. The lowest and outermost point at which the heartbeat is felt is slightly beyond the normal point, although by percussion enlargement may not be determined. Premature contractions are occasionally present and sinus arrhythmia is frequent but insignificant since it often occurs in healthy children. The electrocardiogram may show increased conduction time. On auscultation, a soft blowing systolic murmur is heard at the apex. It may be heard over the whole precordium, or

solely at the apex or even at the pulmonic area only. Such a murmur may be erroneously interrupted as functional if it is localized, *i.e.*, not transmitted in any direction, and if due attention is not paid to the whole picture. An important point to take into consideration is the presence or absence of fever. A persistent slight rise of temperature to 100° F. is as significant when associated with the above described symptoms as a rise to 101° F. or more. Almost equally important is loss of weight. During the active phase of cardiac rheumatism it is common for the child to complain of epigastric pain which is probably pericardial, not gastric in origin. Precordial pain is extremely significant, and is sometimes the only complaint of a child who subsequently develops definite signs of cardiac rheumatism. In such a case a very careful search should be made for rheumatic nodules.

The following case shows the difficulty in obtaining adequate care for a child with a mild carditis. The child, a girl of three years, attended a nursery school and was, therefore, under close observation both by the teacher and the physician. A faint systolic murmur was noted on one of the regular examinations, but was at first thought to be purely functional in character because of the child's age and absence of any other signs. Soon after the teacher noticed that the child was less active physically than before, and cared more for sitting still than playing with the other children. She lost a little weight, and her appetite was less good than formerly. About this time it was found that the child was running a constant slight fever, 100° F. or a little over, and the murmur was rather louder though still faint. No definite enlargement of the heart could be demonstrated, and unfortunately x-ray examination was not possible. No other cause could be found to account for the fever, and one very small nodule was found over the right elbow. The diagnosis was then clear and the mother was told that the child was ill and required special care. She was a young, not very intelligent Irish woman and, although every attempt was made to explain to her tactfully what the real condition of the child was, she refused to believe anything was wrong. She was unable to care for the child at home, as she worked herself, and she had the very common fear of hospitals. When we insisted that she do something about it, she took the child to a physician who had never seen her before and evidently had no special interest in or knowledge of heart disease in childhood, and who told the mother what she wanted to hear, namely, that the child was perfectly well. Since the mother had paid for this opinion, it had more weight with her than

the other, with the result that it was impossible to get any coöperation from her in the attempt to care for the child and prevent bad damage to the heart. For months the child went from pillar to post, until she finally reached a state where the condition was readily diagnosed and confirmed by other physicians and clinics to which the child was taken. Such cases are discouraging to the physician who wishes above all things to prevent serious heart disease. Early diagnosis and the appreciation of the meaning of certain conditions are as essential as in the diagnosis of incipient tuberculosis. We see many similar cases in the clinics. The child does not complain, and when we explain to the parent that, to prevent serious condition, the child should be kept

CHART III
TEMPERATURE CURVE IN RHEUMATIC CARDITIS



J. M., male, aged 8 years

Diagnosis

1. Enlarged heart
2. Mitral insufficiency
3. Mitral stenosis
4. Pericarditis
5. Many subcutaneous nodules

in bed, the answer almost invariably is, "She won't stay in bed—she will get up and run around. She will be quieter if she goes to school and sits down all day." The answer is, as it is in all public health problems, education of the public, but this is a long and difficult task. We can, meanwhile, simply struggle along as best we can with the individual cases.

This brings us to the cases of carditis which are caused by a more overwhelming infection of the rheumatic organism. The fever is 101° to 102° F. and irregular rather than sustained in type (Chart III). The pulse is rapid, more so than the moderate fever would cause. Respirations are somewhat increased, the child really feels sick and is glad to be in bed. The cheeks are flushed, with perhaps a slightly cyanotic tinge. Precordial pain is frequent. Such bouts occurring

without arthritis or chorea are more frequently second attacks rather than the first. Also, although the carditis may and does occur as the first and only evidence of rheumatic infection, there is usually sooner or later some other manifestation, such as brief involvement of some joint, or the development of chorea during the attack of carditis.

The physical signs and symptoms of a severe attack of carditis will not be described here, as it will be covered in a later chapter. It must be emphasized, however, that in children the term "carditis" and "decompensation" go hand in hand. The heart which is actively infected with the rheumatic organism is a failing heart. The symptoms of decompensation are present—pallor, cyanosis, rapid pulse and respiration, and dyspnea. With this type of failure may go the symptoms of congestive failure, enlarged liver, edema, ascites, etc. But when a child is in this latter state he is practically always suffering at the same time from an active infection as evidenced by fever, increased leukocyte count, fleeting or persistent arthritis, and precordial pain. This conception of carditis as being the heart-failure of childhood is very important.

RECURRENT SORE-THROAT AND TONSILLITIS

Because of the frequency with which children with rheumatic heart disease have a history of recurrent attacks of sore-throat or tonsillitis, and the regularity with which the pharynx is inflamed during an attack of rheumatism, the tonsils have been considered the focus of infection. Tonsils have been and still are removed in wholesale fashion from the throats of cardiacs and potential cardiacs for this reason. Whether this is a wholly wise procedure is open to discussion, and will be considered in a later chapter.

The tonsillitis caused by the rheumatic organism does not differ clinically from an ordinary attack. There is fever, painful throat, rapid pulse and often cervical adenitis. The association of these symptoms with definite rheumatic manifestations makes the rheumatic origin of the tonsillitis probable. A history of such attacks in a child with heart disease, but with no other rheumatic history, leads to the assumption that the throat was the avenue through which the heart was infected.

The treatment of a tonsillitis of rheumatic origin is the same as that of any attack. However, if the child already has heart disease, he should be handled as if he were sicker than he really is. He should be kept quietly in bed until completely over the attack, and for several days thereafter in order to spare the heart as much as possible.

At present it is not possible to differentiate a rheumatic tonsillitis or pharyngitis from any other type. If the researches of Small or Birkhaug, or any other investigator, are confirmed, we will be able to determine by throat cultures the presence of the rheumatic organism, and treat the case accordingly.

The British studies on social conditions and acute rheumatism include a survey of this question. These investigators found that "the proportion of healthy throats in the control series of non-rheumatic patients is definitely greater than in the rheumatic patients; this confirms the general belief that a close association exists between rheumatism and sore throat in children. . . . In the St. Thomas's Hospital data, the proportion of healthy throats in the children belonging to the non-rheumatic families is significantly higher than in the members comprising the rheumatic families. A like but smaller difference exists in the corresponding groups in the Glasgow data, but it cannot be considered to be statistically significant. The incidence of healthy throats is not significantly higher in the members of the control families than in the non-rheumatic members of the rheumatic families.

" . . . Among 907 rheumatic members of rheumatic families, 295, or 32.5 per cent, had had operations for removal of the tonsils. One hundred twenty-nine, or 43.7 per cent, of those who had undergone operation developed rheumatism at some period after the operation. In 60 cases, or 46.5 per cent, of those operated upon before the onset of rheumatism, the tonsils had been completely removed, so that operation in these cases failed to protect the children against attack by the disease. It is curious to note the preponderance of attack by chorea over other forms of rheumatic manifestation in those 60 cases whose tonsils had been removed. Sixty-six and seven-tenths per cent of those who developed a first attack of rheumatism some time after the removal of the tonsils had it in the form of chorea, and of these, all but 4 had no other manifestation of the disease. This result suggests that removal of the tonsils affords no protection against attack by chorea."

Although there is a very definite improvement in the incidence of colds and sore-throats following tonsillectomy, the results found by most observers on the effect of the incidence of rheumatism have been discouraging. Kaiser compared the results of tonsillectomy in twenty thousand children with twenty-eight thousand in whom the tonsils were still present. He concluded, in general, although his percentages did not show wide differences, that the tonsillectomized child has a

better chance of escaping rheumatic infection than a non-tonsillectomized child.

Of 194 children who had had rheumatism, observed in the Bellevue outpatient department for periods of one to eight years, about 28 per cent improved, 68 per cent showed no improvement and the remainder grew definitely worse following tonsillectomy. The point may be that if tonsils are removed before they become infected, or before the infection becomes systemic (and this certainly means before the children reach school age), the protection afforded is greater than if the tonsillectomy is performed after infection has taken place.

When the child is already infected with rheumatism, each case should be studied individually before operation is advised. Now and then the operation stirs up the infection so that the child immediately develops chorea, rheumatic fever, or endocarditis, as a direct consequence of the tonsillectomy, which presumably throws the infecting organism into the blood stream. In a child with well-established heart disease whose tonsils have been infected but are now chiefly scar tissue, the wisest course probably is not to operate.

An extremely interesting point found by all investigators is the fact that the effect of tonsillectomy on the occurrence or recurrence of chorea is practically nil. This lends support to the suggestion that some cases of chorea may not be of infective origin, but may be better explained by the fact that the tonsils are only one of the portals of entry all of which cannot be removed. The importance of the accessory nasal sinuses as foci of infection must not be overlooked. It is possible that the infection may smolder in the lymphnodes as in tuberculosis.

SUBCUTANEOUS RHEUMATIC NODULES

These nodules may occur in conjunction with any of the other forms of rheumatic fever, and are generally considered to be evidence of a severe chronic infection. They are found in the deep fascia over bony prominences, and in any tendon. They are most frequently located over the olecranon processes, scalp, vertebræ and patellæ, and may also be found on the feet and hands (Figs. 17, 18, 19). In one case of heart disease with pericarditis, the first nodule was noted on the chest directly over the apex of the heart. A few days later crops appeared on the knees, elbows, scalp, feet and hands. The very small nodules are best felt with the skin loose, and may feel like a very small seed which can be rolled freely beneath the skin. The larger nodules are easily seen when the joint is bent and the skin

stretched tightly over them. The consistency on palpation is fairly firm, the skin moves easily over the nodule. Usually there is no pain or tenderness connected with the nodule, but in exceptional cases this may be so. It has been thought that nodules are more frequently found in England than in this country, and are even spoken of as rare in America. If they are searched for carefully in all cases of heart disease, rheumatic polyarthritis and chorea, they will be found to be not so rare as is commonly thought. Outpatient-department cases should be particularly carefully examined for these nodules as they have a distinct prognostic significance and are always rheumatic in origin. Merritt

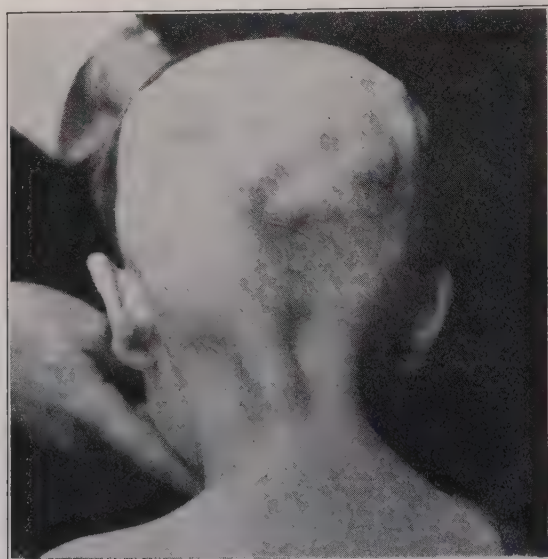


FIG. 17.—SUBCUTANEOUS RHEUMATIC NODULES IN BOY WITH SEVERE CHOREA AND CARDITIS. SCALP.

examined 224 children admitted to Bellevue Hospital with chronic cardiac valvular disease, chorea or rheumatism in the period 1924 to 1927. She found that a total of twenty-four different children, or 12 per cent, had nodules. Wilson found 14.5 per cent in her series, and Poynton 19 per cent. The most striking point in Merritt's study was that in the year 1924-1925 there were sixteen cases with nodules, while the two succeeding years each had only six cases. This higher incidence for 1924-1925 was noted by other observers in this country and in England also. She included only cases where there was no possible doubt of the diagnosis and the figures are therefore con-

servative. They occurred most frequently in children with severe cardiac involvement. Coates, in an examination of one hundred children attending an outpatient department in Bristol, found six children with nodules and other rheumatic manifestation; five had nodules before any other rheumatic manifestations occurred, and seven had nodules plus other rheumatic manifestations. He also states in another article that many children with a latent rheumatic infection show nodules without a definite heart lesion. These nodules may be very



FIG. 18.—SUBCUTANEOUS RHEUMATIC NODULES IN BOY WITH SEVERE CHOREA AND CARDITIS. HAND.

(K. Merritt, *Am. J. Dis. Child.*, Chicago, 1928.)



FIG. 19.—SUBCUTANEOUS RHEUMATIC NODULES IN BOY WITH SEVERE CHOREA AND CARDITIS. ELBOW.

(K. Merritt, *Am. J. Dis. Child.*, Chicago, 1928.)

small, but show the characteristic microscopic picture. Merritt feels that this is contrary to her findings, since in her series there was only one child with rheumatic nodules who had no cardiac lesion. Since her series was among hospital cases only and she included cases in which nodules were undoubtedly present, it cannot properly be compared with Coates's findings in ambulatory cases. It is generally felt that the presence of subcutaneous nodules indicates a severe, chronic process and that sooner or later the heart will become severely damaged.

Although rheumatic nodules are more often found during second attacks of rheumatism and when the heart has already been permanently damaged, they may occur during an attack of chorea. One

child, a boy, was admitted to Bellevue Hospital with an attack of chorea of the type which simulates a paralysis. After a sojourn in the hospital of several weeks with no improvement of the chorea the first nodules were found. He subsequently developed many very large nodules scattered over his body, most numerous in the scalp.

The nodules may last only two or three weeks, or may persist, growing gradually smaller, for months. They do not require treatment for themselves, but their presence means that the patient needs treatment designed particularly to spare the heart.

GROWING PAINS

The term "growing pains" refers to the fleeting joint and muscle pains of which children so frequently complain. They may be momentary in character and occur in any part of the body, although they are most common in the extremities, or they may persist for several hours. Leg ache from overfatigue may be confused with real rheumatic pains. The danger lies in overlooking the more serious condition and putting it down as fatigue. Sometimes the child complains of a knee or ankle so painful that he is unable to walk for a short time after arising in the morning, or at bedtime. The pain is not sufficiently acute to dignify by the term acute rheumatic fever, particularly as there is no rise in temperature. Undoubtedly not all of the so-called growing pains are rheumatic in origin and sometimes they occur in connection with a mild sore-throat or with dental infection. Bad posture and flat feet frequently cause pains in the feet, ankles and even thighs, hips and lower back. On the other hand, some patients with rheumatic heart disease give a history of growing pains as the only previous rheumatic manifestation, and in such cases they are probably rheumatic. It is not enough to prescribe salicylates to relieve the pains, providing they are persistent enough to bring the child to the doctor. The diagnosis must be made, and this requires conscientious observation. The heart should be carefully observed at regular intervals, the child's life and environment should be studied, and the diet and hygiene improved in an attempt to prevent the development of a serious condition.

PURPURA

Various skin lesions may occur during the course of an attack of rheumatic infection. There may be an extensive mottling and flushing of the skin, there may be simply an erythema on the palms of the

hands, or the so-called erythema multiforme may be present. Erythema nodosum has been thought to be always rheumatic in origin, but recently it has been held to be tuberculous in nature. A ten-year-old girl, recently a patient in Bellevue Hospital, was admitted for painful lesions on the extremities, associated with fever. On examination she was found to have numerous round, hot, red, elevated lesions on the legs and thighs and several on the arms. The size varied, but they were all exquisitely tender to palpation. The temperature was 103° F. The child gave a history of acute rheumatic fever in the past, and she had a definite double mitral lesion of the heart. Her five-year-old brother is just beginning his first attack of chorea. The erythema nodosum subsided very promptly on treatment for an acute rheumatic infection. The Mantoux test was negative. This case seems clearly to be purely rheumatic and not tuberculous, since the child had other indubitable rheumatic lesions and there was no evidence of tuberculosis elsewhere in the body. (There may be more than one type of the disease.)

That this reasoning may not lead to accurate conclusions is shown by the case which Coombs cites of pericarditis and lesions of erythema nodosum which proved to be tuberculous in nature.

CHAPTER VI

METHOD OF STUDY

HISTORY

In heart disease perhaps more than in other conditions, a careful systematic method of study must be followed if serious error is to be avoided. The history, physical examination, x-ray, electrocardiogram, all give us valuable information. It is desirable that a systematic plan be carried in the mind of the observer from the time the patient first comes under observation, no matter whether he has rheumatism, chorea or other predisposing disease, or beginning signs suggesting cardiac involvement. Unless such a plan of study is kept in mind the observer may forget to make important observations, which he may regret deeply at some later time, for the lack of a complete record often obscures not only the diagnosis but also the prognosis. For instance, a child is seen at one time with a murmur suggesting mitral disease. At a subsequent examination the signs have changed and aortic disease seems more probable. If carefully recorded observations are kept, the appearance of a new lesion is noted early, signs of failing heart muscle are noted at their first appearance and the child's life may be saved by early treatment.

The history frequently is of prime importance in making a diagnosis. It should be complete in every detail of the past life of the child, not only in regard to the heart and related diseases but should inquire into all of the facts which concern the child as a child, not merely as a cardiac patient. In taking the history it is tactful to get first the facts concerning the condition for which the child is brought to the physician before those from the parents' point of view apparently not important. Parents are frequently irritated by a physician who forgets this. They see no reason why a birth history, family history, past history, diet, habits, and so forth, should be elicited when they are anxious about the present condition only.

Family History.—The family history has a good deal more bearing than seems apparent at first thought. Rheumatic heart disease is probably not hereditary, but the tendency to rheumatic infections runs

in some families. This may be environment, since people living together are exposed to the same factors which cause disease. One cannot study the incidence of disease in large numbers of families without feeling strongly that there is a definite tendency for certain families to have one set of diseases, while others will have an entirely different set. Body type, which is largely hereditary, has been shown to be associated with disease in unmistakable fashion. There is no doubt that rheumatic infections are frequently seen in more than one member of a family. We have seen many examples of two or three children in one family with heart disease or other rheumatic conditions and this has been noted in other cardiac classes.

In a doubtful case it is, therefore, of some help to go carefully into the history of all the members of the family, because a definite story of rheumatic fever or heart disease in one child makes it more likely that another doubtful case is rheumatic.

This association may be due to direct infection from one child to another or from a common source. An example of the latter was a family in which two members were taken with acute rheumatic fever simultaneously. They had been together in New York for two or three days, then one went to Boston, one to New Jersey. Twenty-four hours after they had separated both were attacked with acute rheumatic fever.

Past History.—The past history must be divided into two parts, first the history which should be taken about every child's life in order to get a complete picture of his background, and second, the history directly related to the heart and to rheumatic fever.

The *birth history* may not be important from the cardiac standpoint, but must not be forgotten, for it affects the condition of the individual. A bad start due to difficult labor, prematurity, small size at birth, early feeding difficulty, may prepare the soil which the rheumatic virus finds fertile in later childhood. The condition of the infant at and after birth is of extreme importance. Cyanosis is the first thing about which careful inquiry must be made. This may have been due to asphyxia, or intracranial hemorrhage, to other abnormalities or to congenital heart disease. The symptoms which point to these should not be forgotten. Was the baby cyanosed deeply at birth? If so, how long did the cyanosis last? If not seen at birth, when was it first noted? Was it constant or intermittent? Was it brought on by crying or improved by the deep breathing necessary for a loud cry? The character of the first cry, and whether it was easy to make the baby cry, must

be ascertained. The character of the breathing, whether the baby breathed normally or required vigorous methods of resuscitation after labor, and whether it was necessary to stimulate breathing later, are important. The way the baby nursed in the first few hours and days of life is frequently overlooked in taking histories. This is a fundamental symptom because the abolition of the normal sucking reflex is a sign of the most serious importance.

The most common condition which may cause cyanosis, easily confused with cyanosis due to congenital malfunction of the heart, is intracranial hemorrhage in the newly born. In a considerable percentage of all births there is a tentorial tear or other intracranial injury, even without fractures of the skull, with resulting hemorrhage in the subdural space. There may be hemorrhage into the brain substance, although this is less frequent. These hemorrhages may be large enough to cause early death or severe symptoms, or they may be compatible with life and give few or no symptoms. These children frequently cry and breathe poorly and become cyanosed either constantly or intermittently. In this way they suggest congenital heart disease. The mild cases often have no other symptoms. They may cry much or very little. A most suggestive symptom is the character of the nursing. A child who will not suck, who will not take hold of breast or bottle for the first few days must always be suspected of intracranial injury. In our experience this symptom is lacking in congenital heart disease, except possibly in the most severe cases in which the infant is too feeble to nurse. It is easy to be misled by mothers in this regard. The question as to the early nursing may be misunderstood. It is not sufficient to ask, "Did the baby nurse well at first?" Many babies are lazy, sleepy, not interested in food in the first days, but have a definite sucking reflex. Others will nurse a few minutes only, then stop because they have obtained enough from a free-flowing breast. In both these cases the mother may think the baby did not nurse well. It is, therefore, necessary to determine whether the baby would nurse at all before deciding that this fundamental symptom is present. It is difficult or impossible to get accurate information about this from unintelligent mothers, or after a long lapse of time, or from mothers who were extremely prostrated from a difficult labor.

The character of the infant's breathing even in the early weeks may be altered by congenital diseases of the heart. The breathing may be rapid, and panting constantly, or it may be normal only after crying or other exertion.

The baby's *weight curve* is sometimes important, since congenital heart disease frequently affects the infant's ability to gain and grow. When the lesion is severe enough to cause cyanosis the infant is rarely up to normal weight. But even in the cases without cyanosis and when the heart action seems little disturbed, it is frequently impossible to make the baby gain and grow at a normal rate. Such children may be underweight and undersized for the first year, for several years, or for life.

The *feeding history* needs to be elicited with care for all infants, especially the duration and success of breast-feeding and the type of bottle-feeding with its results. In older children a briefer reference may be made to this, but it must not be omitted because it is not fair to blame the malformation of the heart for a child's general condition when difficult feeding in the first year is responsible.

The *development of the child* has a bearing on his later condition. Congenital heart disease often postpones the time when the child first is sitting, standing or walking, or when the head is held up. Even talking may be influenced. The mental development may be slightly affected, but to less extent than the physical. Acquired heart disease is rare in the first years, but does occur as early as eighteen to twenty months and then influences development in the same manner as congenital disease.

The *habits and hygiene* of the child have the greatest bearing on his health, strength, physique, resistance to disease and on the prognosis of any disease present. It is futile to attempt the study of any child without a minute analysis of his mode of life and diet. Such an analysis not only helps understanding of his present condition, but gives an idea of how to proceed with his future treatment. The exact hours of retiring and rising, the time of going to sleep and awakening, the character of the sleep, whether it is sound, light, restless or broken, and the time and duration of the nap or rest period should all be ascertained. His appetite, habits and manner of eating, likes and dislikes, food idiosyncrasy, are all important and must be considered in studying a child.

The Diet.—It is not sufficient to inquire whether the child eats everything or what articles of food are given him. An exact average daily diet with the composition and amounts of each meal, the time at which it is given and how long it takes the child to eat it must be elicited from the parent. Any food taken between meals must be included as well as the time at which it is taken.

The Bowels.—The conscientious careful physician will inquire into the bowel habits, the exact time at which the bowels generally move and the character of the stools. A history which states that the "bowels are regular" is not adequate. This often means that they are moving every twenty-four hours, but at night, which is not the physiological time and may mean relative constipation.

Bad habits, such as thumb-sucking, nail-biting, masturbation, bed-wetting, quarrelsomeness, unmanageableness and irritability, must not be forgotten, since they give an idea of the kind of individual the physician is treating. Children who have some of these habits are often willful, uncoöperative, difficult patients to handle, and promise poor results. The prognosis of heart disease depends very largely on the docility and willingness of the child to coöperate, as well as on the intelligence of both the child and the parents.

The *amount of exercise* and its effect on the child must be carefully studied. With definite heart involvement exercise often causes dyspnea or palpitation, and many mothers will notice fatigue after moderate exercise as an early symptom. The type of child is a factor in this respect. Some children are active and exercise vigorously, while others have to be urged to take part in games involving physical effort. This aspect should be inquired into more carefully under the present history, but it is very important to find out when the mother first noted abnormal symptoms resulting from the amount of exercise to which the child is accustomed.

The *history of past infectious diseases* must be gone into most thoroughly in all children suspected of having heart disease. Measles, whooping-cough, mumps and chickenpox, which practically every child has sooner or later, have little etiological bearing on the rheumatic infections or on the heart itself.

The disease of childhood, not included in the term rheumatic fever, which seems closely related to the onset of heart disease, is scarlet fever. It is not sufficient to ask whether the child has had scarlet fever. It may have been called "scarlatina" by the euphemistic doctor or it may have been unrecognized. Therefore, one should inquire for any disease with sore-throat, vomiting, rash or for peeling after a minor illness. The severity and duration of the attack of scarlet fever and the presence of complications in lymph-nodes, ears, kidneys or the heart itself should also be noted.

The past history of rheumatic fever is, unfortunately, most diffi-

cult to obtain in many instances. It is not sufficient to ask whether the child has "ever had rheumatism." The parents usually have a mental picture of this disease based on the deforming arthritis of some elderly relative or on the pains of neuritis or sciatica. One must ask specifically whether there has ever been an illness attended by pains in the joints or muscles, even without joint swelling, heat or redness. The familiar "growing pains" are often overlooked, or considered unworthy of notice. The subjective symptoms are often so insignificant that both parents and the child must be most carefully questioned before any complaint of pains or aches can be remembered. Very frequently, no history of anything resembling an acute attack can be obtained. When there is a definite history of fever, its extent and duration should be noted, as well as chills and sweating. Some children or parents will note the appearance and disappearance of rheumatic nodules. The frequent antecedent sore-throat or respiratory infection is important, but the absence of this in the history means little. The history of chorea is often uncertain because other twitchings are confused with it. The common habit spasms of childhood are often thought to be chorea, and even the fidgeting and inability to keep still of the restless child must be discounted in taking the history. Real chorea is usually fairly severe and less likely to be overlooked than other forms of rheumatic fever.

Diphtheria and influenza more often affect the heart muscle than the valves, but may cause symptoms or signs of disturbed heart action. Pneumonia and typhoid occasionally also leave a weak heart muscle.

Syphilis very rarely affects the child's heart, but does stunt and retard the individual.

SYMPTOMS OF CARDIAC INVOLVEMENT

There is no phase of heart disease where one must be more on his guard than in the matter of symptomatology. In the first place, as in all medical histories, there must be no leading questions, suggesting the answer expected to the patient, and much less to the parent. Parents who have been told that their children have heart disease are usually extremely apprehensive and are apt to exaggerate the slightest complaint of the child. The child, on the other hand, is not particularly interested in his heart, nor with symptoms which arise from it, and gives heed to them only when they obtrude forcibly upon his consciousness. It is wise, therefore, to ask about pain, palpitation, dyspnea, edema and so forth in a casual way and not to let the parent or

patient suspect that these apparently unrelated symptoms are associated with each other or with the heart. Later it may be necessary to educate both parent and child to watch for these danger signals in an intelligent way. It is our experience that the largest group of patients applying to an outpatient department on account of real or fancied heart disease come because they have been told by the school physician or by a private practitioner that the child has something the matter with the heart. It is an interesting fact that in a large number of these cases no sign or symptom can be elicited by careful and repeated examination. Perhaps some of them are sent for diagnosis when the physician is in doubt. We suspect that in the case of the private practitioner the transient murmur heard during an acute febrile illness is commonly mistaken for an organic lesion. Another large group is composed of the children who have a soft functional blow at the apex or base, without real meaning. It is relatively rare to have a child brought for symptoms referable to the heart. Even the true organic cases often are discovered by accident and have noted no symptoms.

Symptoms of Carditis.—In the hospital ward the early cases in which carditis follows polyarthritis or chorea usually have no discomfort, and no heart disease is suspected. Pericarditis occurring early is, of course, an exception and does give pain. Congenital heart disease will be considered separately.

The symptoms which really do occur are the same seen in adults, but, as a rule, only come on after the heart disease has been in existence for some time.

The child whose heart is involved after rheumatic arthritis or chorea often remains anemic and does not gain weight, becoming undernourished unless he has been unusually plump. Thus, a great many mothers bring their children because they are pale and thin and generally "run-down." This is the most common complaint in cases where we are the first to discover heart disease. Such a child may have a mitral valve which is considerably damaged, with some cardiac enlargement and a loud murmur, yet no pain, palpitation or dyspnea have ever been noted. But let the mother once know that there is heart disease and what cardiac symptoms are, and she will nearly always bring a tale of pain and palpitation, or extreme shortness of breath after a few weeks of worry over the child.

The layman, and sometimes the physician, thinks first of pain

when the symptoms of heart disease are mentioned. Pain, of course, is the least complaint of the cardiac child and occurs most often in pericarditis or severe decompensation.

In the more severe cases of rheumatic fever or chorea followed by rapid cardiac involvement there are more likely to be symptoms. But again they are often not cardiac symptoms. The rapidly developing anemia and loss of strength make *weakness* and general disinclination to get up or to do anything an early symptom. The appetite often fails, the child is languid, pale, chronically sick in behavior and appearance. If he gets out of bed, he lies around and does not feel like playing actively. An examination at this stage may show a heart which is acting somewhat poorly, a fully developed valvular murmur and an electrocardiogram showing definite heart-muscle involvement.

Eventually the muscular involvement makes itself felt and true cardiac symptoms appear.

Dyspnea is one of the first symptoms, slight shortness of breath on exertion, such as going upstairs, or even getting up and about the room in severe cases. It is remarkable how much dyspnea is borne by the child before he complains of it. He may be definitely panting after exercise or even in bed, but says he can breathe all right. The child whose heart involvement is slight at first, may go on for months before dyspnea is noted. It is very easy to be misled about this question of dyspnea. If the mother or child is asked whether the latter gets out of breath on going upstairs, the answer is frequently in the affirmative, but closer questioning brings out the fact that he takes three to five flights without stopping and even then the dyspnea is no more than normal for any individual.

More severe cases do get short of breath on stairs or running, soon find that they cannot run at all and have to pause on every flight. Unfortunately, many who should stop on this warning fail to do so, cannot be made to do so, and go on overtaxing their hearts because the desire for play is so deep-rooted.

The degree of this dyspnea increases with the seriousness of the lesion until we come to the decompensated patient who has dyspnea at rest. This may be slight, shown by a moderate acceleration and deepening of the respiration, or, finally, it may become a struggle for breath, pitiful to see.

The severer cases breathe more easily in the erect position, propped up with pillows, a back rest or sitting in a chair. This semi-erect posi-

tion definitely must throw more work on the circulation than the horizontal one, but this is compensated for by the greater ease in using accessory muscles of respiration. Strange to say the onset of pneumonia often makes a cardiac patient who has been orthopneic perfectly comfortable when horizontal. We frequently see such a case, who has been sitting up in bed for weeks, lie flat through the pneumonia and have to sit upright as soon as the acute disease is past. The increased rate of respiration seen in pneumonia may account for this.

Disturbed heart action as a subjective symptom is less frequent than one would suppose. Heart disease must be fairly well advanced to make the heart act badly enough to become noticed by the child. It is only in the moderately severe cases that the overaction after exercise becomes noticeable, and even then it does not seem to distress children very much. The child's failure to realize the importance of disturbed heart action is probably a factor, so that he disregards a palpitation or arrhythmia which would distress an adult, partly from its psychological effect.

The failing heart with its tumultuous irregular action is distressing to the patient, but the child often refuses to admit any discomfort. Paroxysmal tachycardia, which occurs rarely in older children, may distress the child, but sinus arrhythmia, premature systoles, even fibrillation, are usually not noticed.

Pain does occur in the severe cases but it is not often very extreme and is generally due to pericarditis. Even an acute fibrinous pericarditis may be associated with relatively little pain.

The symptoms presented by the child with total loss of cardiac reserve, with no exercise tolerance, are dyspnea and disturbed heart action and further symptoms of failing circulation.

Edema comes relatively late. It is seen in the legs first, then about the genitals and the lower abdominal wall.

Ascites occurs in severe cases eventually and is a serious matter. The abdomen may contain a good deal of fluid before it troubles the patient.

Digestive disturbances are not uncommon in heart disease. Loss of appetite may occur early, as in any illness acute or chronic. Subjective symptoms of indigestion are rarely seen in children. Vomiting comes on when there is congestive failure and may be due to the failing heart and congested viscera, not to the digitalis which is being

administered. Epigastric pain may be present and occasionally severe abdominal pain which simulates acute appendicitis.

Cerebral congestion may cause insomnia, restlessness, even delirium.

Vascular accidents are seen fairly often with heart disease. These are most often cerebral, due to emboli from the vegetations on the valves or from auricular thrombi. The result is usually a monoplegia or hemiplegia with aphasia if the lesion is on the speech center side. The onset is sudden in children with moderate or severe heart involvement. The course is slow and recovery not often complete, although the residual symptoms are nearly always less than the initial ones.

Emboli thrown off into the circulation are much less common in rheumatic heart disease than in subacute bacterial endocarditis. The explanation lies in the fact that the vegetations on the valves of the rheumatic heart are closely adherent and much less friable than those found in subacute bacterial endocarditis. *Thrombosis* of the vein may occur in rheumatic fever, usually in the upper extremities, but is rare. Two cases occurred among the children in the wards at Bellevue in a few months. In one case the right and in the other the left forearm was involved. Both were desperately ill children who died soon after the development of this symptom. These children both had pain about the elbow followed by swelling and edema of the forearm and hand, caused by obstruction in the venous circulation. Such a condition is best explained by a mural thrombus formation. Although the histopathology of the vein has not been studied as carefully as the arteries it is quite likely that there is a rheumatic inflammation of the vein walls which could cause a thrombus formation.

In contrast to this is the frequency with which embolic phenomena occur in subacute bacterial endocarditis. In this disease emboli broken off from the heart valves may lodge in any artery.

If an embolus lodges in the brachial artery the radial pulse will either be absent or much smaller than in the other arm. The patient will complain of pain at the site of lodgment and a mass may be felt in the artery. The chief danger lies in possible failure of collateral circulation to be established. If this occurs, gangrene follows in the area which is deprived of its blood. In a case of subacute bacterial endocarditis (described elsewhere) such an embolism occurred. Within a week an aneurysm formed in the brachial artery above the site of the embolus, due to the weakened wall of the artery plus the burden put on it by the embolus. In this case the embolism occurred at the bifur-

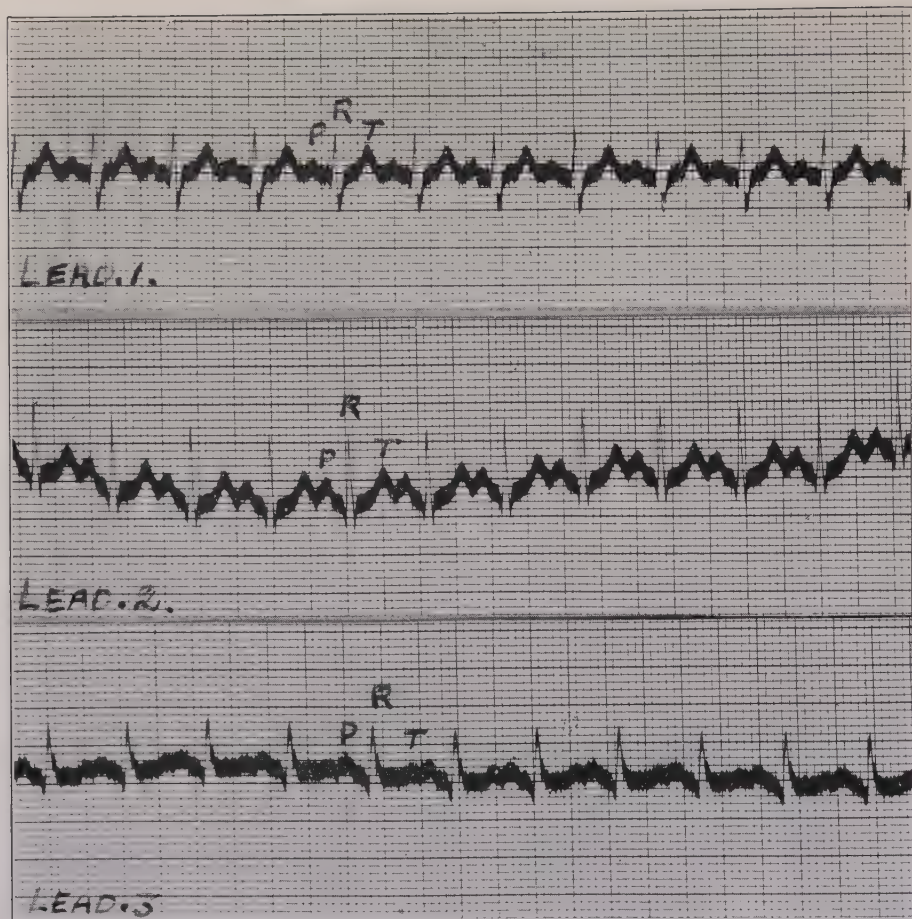


FIG. 20.—EFFORT SYNDROME.

Same patient as in Figure 21. Electrocardiogram shows simple tachycardia.

cation of the artery, with partial obstruction to the radial and ulnar arteries.

Effort Syndrome.—This term came into general use during the war for a class of functional heart cases which then occurred for the first time in large numbers. These patients had no organic heart disease but an exaggerated response to physical exertion, mental effort or emotional stress.

The signs and symptoms are out of all proportion to the energy expended and are as follows: Easy fatigability, faintness, tremulousness, palpitation, heart pain, abnormal dyspnea on exertion, and tachycardia. The apex-beat is often vigorous. Accidental murmurs may be present.

Effort syndrome may be found in an individual who has real heart disease. In such a case, it will be necessary to distinguish between the symptoms due to neurosis and those due to organic disease of the heart.

In childhood this condition is rarely seen, but something similar does occur in adolescence. Boys at puberty or slightly later, who have grown very rapidly, often have symptoms and signs resembling the "soldier's heart." Some cases come on after acute infections, especially after influenza. These boys have rapid heart action, increased unduly by exercise or emotion. The heart is not enlarged, but may even seem small for the size of the boy. The sounds are normal, there are no murmurs and the blood-pressure is normal. The patient may not be especially conscious of the rapid heart action, though some boys get hypochondriacal over it when it is called to their attention.

The following case illustrates the condition of effort syndrome:

December, 1928. H. A. Jr., sixteen years. At boarding school, working too hard, sleeping and eating too little. Since Thanksgiving had had a cold, slight fever the first few days, slight cough. Cold in head most of the time. Had a purulent nasal discharge. He was $69\frac{3}{4}$ inches tall, weighed 132 pounds (average weight 146 pounds), moderately thin. The heart was normal in size, rapid (130 standing), regular, forcible, sounds normal, no murmurs. After sitting, his pulse was 120. Examination disclosed a nasopharyngitis but no sinusitis. Ten days later the pulse was 140 standing, and the blood-pressure was 125/80; and when sitting, the pulse was 120 to 130, the blood-pressure 130/80. He had no subjective symptoms but was worrying about his heart. The electrocardiogram was normal (Fig. 20). The

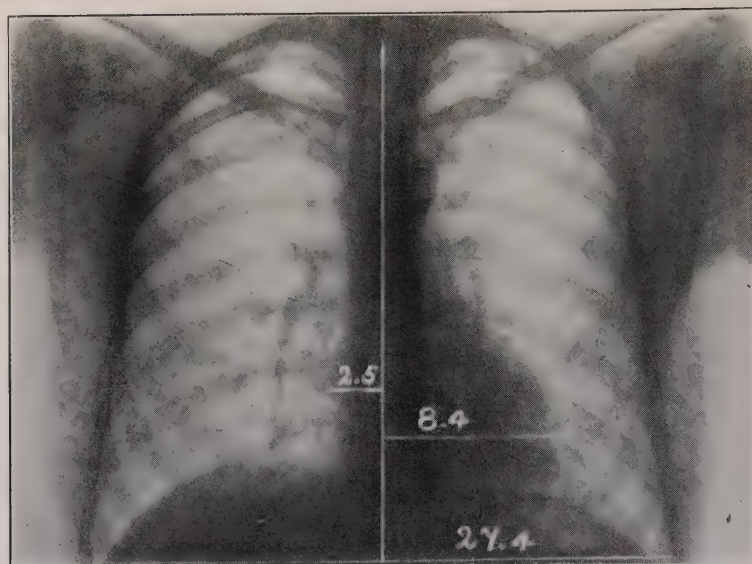


FIG. 21.—EFFORT SYNDROME.

Same patient as in Figure 20. Heart shadow is normal.

x-ray showed a heart somewhat small (Fig. 21). This rapid heart action continued for several weeks, gradually subsided to normal by March.

Another big, rapidly growing boy of fifteen years had a similar condition after influenza. His heart rate was 150 after walking a few blocks. This lasted two or three months, gradually subsiding.

It is not quite accurate to call these cases in overgrown boys' effort syndrome because usually they have no subjective symptoms. They should be spared fatigue and not driven to the routine of a boarding school. Extra rest, feeding, tonics, etc., apparently cure the condition in from one to three months. They should not engage in violent athletics until the heart has had time to catch up to their general physique.

Congestive Heart-Failure.—The severer forms of heart involvement are grouped in Class 3, which is defined by the American Heart Association as follows: "Patients with organic heart disease and with symptoms or signs of heart-failure at rest, unable to carry on any physical activity without discomfort. There is fatigue, palpitation, dyspnea or chest pain at rest. Patients in this class show marked physical signs of congestive heart-failure, or the anginal syndrome or signs of active heart infection."

It is evident that this is a broad group and that the picture does not apply in all its details to every patient. It shades gradually from the cases in Class 2*b* in which there is greatly limited activity and some of the symptoms of failing heart to the cases with real congestive heart-failure.

* The first type of case which one could call Class 3 is the child whose heart is undergoing an acute attack of infection. The heart muscle is enough involved to be unable to carry on the circulation properly at rest without giving symptoms. There is tumultuous or rapid heart action, of which the child is more or less conscious. There may be some pain but this is not common in childhood except in the presence of acute pericarditis. The breathing is somewhat rapid and dyspneic, although he may not complain nor even be conscious of the fact. He is more comfortable sitting up or with the head and shoulders raised than he is when recumbent, though he may be able to lie down to sleep. The heart usually shows signs of valvular involvement, but this may be apparently insufficient to cause the symptoms. As a rule, it is somewhat enlarged.

The damage to the heart muscle is enough to interfere with its action, yet there is no real failure of the circulation.

Such cases may go on to real congestive heart-failure, but the acute process more often gradually subsides, the symptoms gradually disappear and the functional condition must be classified as Class 2*b*. Later such children may be transferred to Class 2*a*, or even to Class 1, for all symptoms may disappear.

It is important for the physician to bear in mind the fact that attacks of cardiac symptoms come on as a result of fresh infection of the myocardium, not from overstrain of the heart due to exertion. Of course it is unwise for a cardiac child to overexert beyond a reasonable point because it is possible for any individual to get up an acute dilatation by extreme exercise. Chronic overstrain or fatigue may predispose to fresh infection also. But in the main, heart-failure comes as a result of fresh rheumatic fever, fresh carditis, with consequent weakening of the myocardium.

The severest group with true congestive heart-failure is made up of children who have usually had several attacks of carditis, or one severe progressive attack. The heart is, therefore, much enlarged and weakened, has one or more definite valvular lesions, and often an acutely inflamed or adherent pericardium. These cases are most commonly those of mitral disease, with or without pericardial involve-

ment, in which the gradually weakening heart muscle results in a relative tricuspid insufficiency. So long as the tricuspid valve is competent the circulation is maintained, even if imperfectly, but once this valve gives way the right ventricle pumps blood back into the great veins. The result is disastrous, since the heart is working against itself, forcing blood in two directions opposed to each other. This is the condition called congestive heart-failure, formerly known as acute or total decompensation. The former is the better term because it indicates the result, *i.e.*, general congestion of the viscera, indeed, of the whole body. Decompensation is a less good term because there is always some decompensation in all patients with any cardiac symptoms, that is, in whom the heart muscle has not wholly compensated for its handicap. The term belongs to the older point of view, in which the damaged valve was the first thought of most medical men and the degree of hypertrophy of the heart muscle to "compensate" for the inefficient valve was considered of prime importance. The modern view is probably more nearly correct, that the heart muscle is always damaged and weakened by the acute rheumatic infection. The poor valve action adds to the burden, but is less important than the myocardial factor.

The symptoms of congestive heart-failure are exaggerations of those mentioned. The dyspnea becomes more and more marked, finally a gasping struggle for breath. The patient is utterly unable to lie down. Hydrothorax increases the difficulty. Palpitation is more distressing and there may be pain of a sticking or aching character. There is a cough, tickling or hacking at first, increasing until it is almost constant, with nearly every breath. The expectoration is mucous at first, becoming frothy and pink with edema of the lungs. It may be quite bloody. The congested liver is tender to touch and even subjectively painful. Ascites increases the abdominal discomfort and dyspnea, while the edema of the legs and of other organs causes discomfort from the tight skin.

The result of this circulatory failure is a congestion of all the organs, congested lungs developing into edema, hydrothorax, enlarged liver, ascites, edema of the extremities, genitals, trunk and even the face and neck. The congestion of the kidneys results in a renal failure which increases the edema.

Many are the physical signs of congestive heart-failure, but all depend on the visceral and somatic congestion and edema.

In the lungs there are râles at the bases, increasing in size and

moisture and gradually involving the whole lung. Sudden attacks of pulmonary edema may come on with pink, frothy sputum, gradually filling up the lungs until the patient practically drowns in his own secretions.

Ascites gives the usual fluid wave and shifting dulness in the abdomen.

Edema is apt to begin in the genitals, especially in males, and extends to the flanks and thighs, more often than to the ankles, since these children are generally sick enough to be in bed before edema begins. If the child is on his feet the ankles are involved first. Facial edema suggests complicating nephritis.

The urine is scanty and contains moderate to large amounts of albumin, some casts, but rarely red or white cells in excess. There is no real kidney failure unless nephritis develops.

True nephritis is uncommon in any stage of rheumatic carditis. The presence of albumin or of casts in the urine is probably an expression of kidney congestion. At autopsy the kidneys are almost never the site of an acute inflammation, so that if nephritis does occur it clears up before death.

CHAPTER VII

THE EXAMINATION OF THE HEART

SYSTEMATIC STUDY

The student of medicine is taught by instructor and textbook to attack the examination of the heart in a definite manner. The facts elicited upon which the diagnosis is to be made are grouped under the method by which they are obtained, *i.e.*, inspection, palpation, percussion and auscultation. This is all very well for the beginner who must be taught the fundamental methods. But we estimate the size of the heart by facts obtained through all four methods. The character of its action is judged by inspection, palpation and auscultation. As a rule, valvular damage is diagnosed by auscultation, but palpation of a thrill may weigh in determining the exact type of murmur present. The result of a description of a diseased heart, which is divided up in this classical fashion, leaves the reader or listener confused and uncertain as to the real condition of the heart described.

Practical experience of physicians working in hospitals and cardiac classes has demonstrated that the best plan is to attack the problem of diagnosis of heart disease from an entirely different angle. Clear thinking and accurate observation follow when one has an idea of what one is trying to determine in examining any heart. In the first place the *size* of the heart is the most important point to be determined, next to the character of its *action*. These facts are of fundamental importance because the function of the heart may be estimated to a considerable degree from them without going further. The condition of the *valves* should be investigated also, because valvular lesions have their importance, although they should not be the first and last thought in studying the heart. The murmur fascinates the tyro, and he is interested in nothing else. Even the trained clinician often forgets to study the heart muscle in puzzling over an increased or obscure murmur.

The examiner should have constantly in mind a scheme of systematic examination of the heart, which is to be used for every case. Such a scheme is followed in the Huddleston Memorial Cardiac Class at Bellevue Hospital.

EXAMINATION OF THE HEART

Size	- Apex impulse - Maximum site - Left limit to which it is palpable * - Character of impulse - Borders - Left - Right	- Percussion (x-ray, at 2 meters)
Action	- Rate of heart - Force - Rhythm	
Sounds	- First - Second - Third	- at apex - at base
Murmurs	1. Time and duration in cardiac cycle (palpate apex, <i>not</i> arteries) 2. Site - Maximum site. - Maximum transmission - Total area heard † 3. Quality, pitch, intensity 4. Effect of posture, exercise, drugs	
Thrills	- Site - Quality	
Pulse	- Strength, quality, rate, rhythm, deficit - Artery wall	
Blood-Pressure	- Effect of posture - Exercise - Mental excitement	

* The left limit of palpability is perhaps the most important single sign. In children this corresponds very accurately to left border and gives size of the heart much more accurately than by percussion.

† Mapping out the total area over which a murmur is audible is extremely important because it prevents overlooking the true maximum site. It is *not* sufficient to listen merely at apex and base for most congenital murmurs, aortic regurgitations, many functional murmurs will be overlooked.

Size.—The size of the heart may best be judged by the *position of the apex*. An apex which is outside the normal site generally means an enlarged heart. Cardiac displacement must be ruled out, however, and this can be done, as a rule, by percussion of the borders and by searching for lesions of the lungs or pleura, which could be the cause of such displacement. In studying the apex the maximum impulse should first be observed by inspection, then by palpation. This is best done with the fingers held flat against the chest wall. It must be remembered that in infants and small children the maximum impulse is well outside the nipple line. It gradually moves inward with the growth of the child until it is in the nipple line at from four to six years. After six years it should be inside the nipple. In the majority of children the maximum impulse is in the fifth space, occasionally in the fourth. Its position depends somewhat on the shape of the thorax.

In the short square type of chest the heart lies more horizontally and the apex is apt to be relatively further out. In the long narrow type the heart is more vertical and the apex may swing well inside the nipple.

More important than the maximum impulse is the lower and outer point of the impulse. This is easily determined in the child on account of the thin chest wall. If the region of the apex is palpated with the *end* of a finger or thumb it is possible in nearly every child to determine accurately the extreme edge of the cardiac impulse. The finger should be moved back and forth, from left to right in an intercostal space until the exact point is found beyond which the impulse is not felt. Percussion and the x-ray will show that this corresponds to the left cardiac border in nearly every instance. This method is easier than percussion and much more accurate in the hands of a person with a poor ear.

The position of the apex is the best guide as to the size of the heart, provided the heart has not been displaced. The most common cause of displacement of the apex is the position of the patient. The whole mediastinum is movable and the force of gravity acts on the heart. A child who has been in bed and lying on one side for some time may have an apex which is considerably displaced. This is not uncommonly seen in a child who has, for instance, acute dry pleurisy. He is more comfortable lying on the affected side and may remain on it for many days. Such a child seen recently had his apex nearly to the left anterior axillary line. He had also a febrile systolic murmur. The presence of a murmur and apparent enlargement was considered good reason for a diagnosis of endocarditis; but when the child was turned on his right side and then on his back the apex assumed its normal position.

Displacement may be due to pleural effusion which causes the apex to move to the opposite side; or to pulmonary fibrosis or pulmonary adhesions which by the contracting of scar tissue pulls the heart toward the affected side. The lungs must, therefore, be carefully examined to see whether there is any extracardiac cause for the abnormal position of the apex.

During this study of the apex the *character* of the impulse is also observed. The normal impulse is circumscribed, felt only over a small area, generally in one interspace, and has a good force. The apex impulse of the hypertrophied heart is usually diffuse, felt over a wider area often in two interspaces; the thrust is more forceful and

sometimes heaving. The dilated weakened heart has an impulse still more diffuse, which may be felt in three spaces and over an area two or more inches in diameter. Its force is poor, sometimes flopping, or wavelike, or it may be reduced to a feeble tap. Study of the apex-beat gives valuable information as to the heart muscle and its functionings.

The *borders* of the heart are determined in the classical way by percussion. The percussed finger should be held parallel to the border being mapped out, not in the interspaces as in lung percussion. It should be held firmly against the chest wall, and the percussion should be firm but not too heavy. The thin chest wall of the child does not require percussion with three fingers. The blow should be quick and the percussed finger should not be "damped" by holding the percussing finger down after striking the blow. The piano-hammer stroke should be imitated. The right border of cardiac dulness is usually at the right sternal border, even though the x-ray may show that the cardiac shadow extends to the right of the sternum. The left border extends obliquely upward from the lower and outer point of the apex impulse to the sternal border at about the second rib. It is not practicable to distinguish between deep and superficial cardiac dulness. In the child the layer of lung which overlaps the heart is thin and narrow, and only in exceptional cases can the two areas be mapped out. In percussing the right border the cardiohepatic angle formed with the upper level of liver dulness should always be noted. Displacement of the right border to the right may be demonstrated by percussion, but when it is displaced only slightly to the left, the sternal dulness often obscures the cardiac outline.

The information required to determine the size of the heart is obtained by all the methods of examination. The apex is located by inspection and palpation and even by auscultation, the borders by percussion, yet the aim is information about the *size* of the heart. When this information is scattered about in several places, as in the first method of description mentioned, it is difficult to obtain a clear mental picture of what the examiner is trying to find out or describe.

Action.—*Rate.*—While the observer has been determining the size of the child's heart he has usually also noted the quality of its action, but if not, this must next be carefully noted. Under heart action the rate, the force and the rhythm are to be considered. The heart rate should be taken by palpating the apex or by auscultation of the heart,

and the pulse rate by feeling the radial or carotid pulse. It must be remembered that the rate is more rapid in children than in adults. A very rapid rate in an infant may have no serious meaning, even if it is 180 to 200. The presence of fever, 104° F. and higher, often raises the heart rate to 150 to 160, without any evidence that the child is in poor condition.

Force.—This is judged by the strength of the apex-beat, by the heart-sounds and to some extent, by the pulse. Normally, the apex impulse has a fairly strong thrust, over an area $\frac{1}{2}$ to 1 inch in diameter, in one or at most two spaces, fifth, or fourth and fifth. There is considerable normal variation from a barely palpable tap to moderately vigorous thrusts. The apex should be felt with the finger tips, or by laying the hand flat on the chest over the heart. After a little experience a good deal of information is obtained by this method. It might almost be said that one obtains more idea of the heart muscle in this way than by any other. Until some functional test is devised which will gauge accurately the reaction of the heart to exertion, the type of study indicated here will give more information about the actual condition of the heart than any other kind of observation at present in use.

A poor myocardium is indicated by a feeble impulse or by one which is flopping, wavelike and variable in force. The action may be overforcible and tumultuous, the apex widely palpable and in the nature of a strong forcible thrust, as in aortic stenosis where there is great hypertrophy of the heart muscle.

Rhythm.—The rhythm of the heartbeat is now realized to be of great importance since the modern study of the heart has disclosed so many abnormal types. The arhythmias will be discussed elsewhere more fully. Their recognition is relatively easy in childhood because children have fewer varieties than adults, although nearly all may appear in the child who is approaching adolescence. Most arhythmias, *e.g.*, premature contraction, auricular fibrillation, second degree heart-block, sinus arhythmia, dropped beats, complete heart-block, can be recognized by ordinary physical examination, while others, for instance first degree heart-block or differentiation between auricular and ventricular extrasystoles, require the electrocardiogram for diagnosis.

In the study of the heart's rhythm by physical examination alone, auscultation is the safest guide. Palpation of the pulse or apex-beat is unreliable. Sinus arhythmia is physiological in a large number of

children and is diagnosed by the acceleration of the heart rate during inspiration.

Premature contractions are not infrequent in normal children. The premature beat follows closely the normal systole and is succeeded by a long pause. The next beat is more forcible.

Auricular fibrillation, which is evidence of failure of the auricles, occurs in older children who present the picture of heart-failure as it appears in adults. It is evident by a totally irregular heart or pulse rhythm. The auricles may beat at the rate of 170 or more per minute while the ventricles assume their own rhythm of less than 100. Sinus arrhythmia plus premature contractions frequently simulates auricular fibrillation. Usually there is a pulse deficit in auricular fibrillation, *i.e.*, the heart rate is appreciably faster than the pulse rate. This is accurately determined by counting the heart rate by means of the stethoscope placed over the apex at the same time that the radial pulse is counted. Two observers are necessary for this procedure. When there is a slow auricular fibrillation with no pulse deficit the electrocardiogram will make the diagnosis. Clinically, auricular fibrillation cannot be definitely distinguished from ventricular fibrillation, which is invariably fatal, but is clearly shown by the electrocardiogram. Ventricular fibrillation is a result of a huge overdose of digitalis and will be further discussed in the chapter on treatment.

Sounds.—*First and Second Sounds.*—The intensity and quality of the sounds should be noted at the apex over the body of the heart and over the pulmonic and aortic areas. In infancy the first and second sounds approach each other in quality, the first being short and more valvular than in later life. The first sound at the apex is normally longer and lower pitched than the second. The latter is less sharp than at the base. The diastolic pause is relatively short in childhood and may be no longer than the interval between the first and second sounds in the infant or with rapid heart action. In this case it is sometimes impossible to tell the sounds apart at the apex, and to recognize the first sound the finger must be applied to the apex alongside the stethoscope. It is impossible to time the heart-sounds accurately by feeling of the radial pulse, because of the lag of the beat at a distance from the heart. The carotid impulse is retarded slightly, and even this delay may be enough to confuse the observer in the case of the rapid heart of a child. The beginner often has difficulty, even when timing the beat with the finger at the apex, because his nervous system is not trained to synchronize tactile and auditory sensations.

This method of timing the heart-sounds by placing the finger on the apex-beat is extremely important because it is the first step in timing murmurs.

At the base the second sound is louder than the first, and (up to five or six years) the second pulmonic is louder than the aortic second sound in the majority of children.

Accentuation of Sounds.—When a sound is louder than normal, it is said to be accentuated. To judge this, the examiner must keep a mental standard and try to remember how loud the two sounds should be at apex and base. Accentuation is more commonly heard in the second sound and at the base. Increase in the blood-pressure in the peripheral circulation accentuates the aortic second sound, as seen in increased blood-pressure of nephritis. Increase in the pressure in the pulmonary circulation gives an accentuated second pulmonic sound as in mitral disease. The large left auricle may push away the lung enough to make the second pulmonic sound louder. The relative loudness of the two second sounds should be noted, but it must not be forgotten that in childhood the second pulmonic is very often somewhat louder than the aortic second sound. Hence an aortic sound equal to or slightly louder than the pulmonic must be considered as accentuated. The pulmonic second sound must not be considered accentuated, unless it is considerably louder than the aortic second sound.

The first sound is not accentuated except in mitral stenosis when it becomes short, loud and snappy.

Diminution of Sounds.—Definite diminution of the sounds is always important. The first sound is often diminished in heart disease, or even entirely absent, and is frequently replaced by a systolic murmur. Rare cases are seen in which there is complete silence during systole, without a murmur.

Weakening or absence of the second aortic sound is found when the aortic valve is stenosed and does not close with a snap. The pulmonic second is feeble or absent in pulmonic stenosis, because the obstruction keeps the blood back from the pulmonary artery and results in a low pressure in it.

Muffling or absence of all sounds suggests pericardial effusion. It is one of the most suggestive signs of this condition, yet is often overlooked, especially by those unused to examining children in whom the sounds are normally louder than those of adults who have thick chest walls. Myocarditis also often results in feeble muscle sounds,

particularly if a great amount of the heart muscle has been damaged. The third heart-sound may sometimes be made to disappear by exercise or by change of position, or it may be induced by the same measures.

Third Heart-Sound.—In many children this is heard normally, but it also appears in pathological conditions. It is best heard just inside the apex, as a rule, in mid-diastole. The sound has a valvular quality not unlike the second sound as heard at the apex. The mistake is often made of calling this a split second sound at the apex, but if the second sound is split, it is split at the base also. Therefore a split second sound at the apex, not heard at the base, means a second sound followed by a third sound. This sound has various explanations, none of which is satisfactory. The most plausible is the sudden stretching of the chordæ tendineæ of the mitral valve by the blood rushing through the mitral orifice swirling behind the valves and tensing the chordæ before the ventricle contracts.

“Gallop Sound.”—The three heart-sounds often sound like a galloping horse and may be the sounds heard in the so-called “gallop rhythm.” This is not a change in rhythm and should be called “gallop sound” or simply “gallop.”

Murmurs.—The presence of abnormal sounds or murmurs is a matter which fascinates the medical student and, indeed, many older physicians. When a murmur is heard all other signs in the heart are too often disregarded. It is evident that the murmur is much less important than the character of the heart action, the quality of the heart muscle, and the efficiency with which the circulation is being kept up. The murmur is so obvious and distracting that these other facts which give more valuable information are too often relegated to the background.

The murmur must not be totally disregarded, however, for it is largely from this physical sign that disease or abnormality of the endocardium and pericardium is diagnosed. Therefore, a careful study gives information which is valuable if properly weighed in conjunction with other findings.

There are many facts about each murmur which must be noted. It is not sufficient to note the time of the murmur and its presence at apex and base. The following systematic study of every murmur is necessary to make it of use in diagnosing the meaning and origin of the abnormal sounds:

- 1 (a) Time
(b) Duration in cardiac cycle
- 2 (a) Intensity
(b) Pitch
(c) Quality
- 3 (a) Site, total area over which the murmur is heard
(b) Maximum site
(c) Maximum transmission
- 4 (a) Effect of posture
(b) Exercise
(c) Drugs

Time.—The time of the murmur is the first and most important point to determine. The cardiac murmurs are definitely separated into two groups, systolic and diastolic. Unless the time of the murmur is accurately determined, no diagnosis can be correct. Here again the only safe method is to put one finger on the apex and relate the murmur to the systolic thrust of the heart, for the one sure and almost unalterable fact is that the apical thrust means ventricular systole. It is futile to attempt to time murmurs by the pulse for the reasons which have been pointed out in the discussion of the heart-sounds. The heart-sounds are also unreliable guides for two reasons. In children, and especially in small infants, the character of the first sound is very much like the second, and when the second is accentuated, as it is in many valvular lesions, the two sounds may be completely confused. Furthermore, the first sound is frequently absent, replaced by the murmur. When this is the case the second sound is accentuated, and if this is mistaken for the first sound the systolic murmur is thought to be diastolic. For these reasons accurate timing of the murmur in relation to the systolic thrust of the contracting ventricle is the first and essential point in its study, and should always be ascertained.

Duration.—At the same time the duration of the murmur in relation to the cardiac cycle can be determined. Not all systolic murmurs fill the entire period of systole. Some are early systolic and are separated from the second sound by a definite interval. Others occupy the latter part of systole following the first sound. Diastolic murmurs also vary in time. Aortic regurgitation usually gives a long murmur filling all of diastole. The murmur of mitral stenosis may be mid-diastolic or late diastolic (the presystolic murmur) and may change

its time in the cycle with increase in the heart rate. Pericardial rubs vary in time, may be systolic, diastolic or both, giving a to-and-fro sound. They sometimes occupy late systole and late diastole and seem slightly out of time with the heartbeat.

Intensity.—The intensity or loudness of a murmur varies from a very faint blow or sighing sound heard only with great difficulty in an absolutely quiet room, to a murmur so loud that it may be heard by the ear held several inches from the chest. The patient himself may be able to hear such a murmur. Loudness is not always significant, for early or advanced lesions often give faint murmurs and the diagnosis is most important when disease of the heart is in its early phase. Aortic insufficiency frequently gives a murmur which is so faint as to be almost inaudible, yet the diagnosis of involvement of this valve is of utmost importance both in planning treatment for the patient and in making the prognosis. The aortic murmur is very frequently missed by the use of a poor stethoscope.

Pitch.—The pitch of a cardiac murmur varies, depending on the mechanism of its production, the size and shape of the orifice and the force of the contracting ventricle. The pitch of the sound is really of minor importance.

Quality.—The quality has more meaning and again depends on the manner of production. Wide-open orifices with smooth edges give soft blowing sounds, narrower ones with irregular edges give hissing, harsh or musical sounds. The variations in quality are almost endless; no two murmurs sound exactly alike. The murmurs of early mitral disease are soft, low-pitched, blowing sounds. More advanced ones are harsher, louder, higher, but the quality and loudness give no idea of the amount of regurgitation. Many mitral murmurs are definitely musical, some whining. The latter have been called the “seagull murmurs” from resemblance to the cry of that bird, a high whine, lowering in pitch toward the end. The murmur of aortic regurgitation is often a soft sigh or blow, low or high in pitch. Congenital murmurs are often very harsh, rasping, sometimes hissing, often a combination of indescribably mixed noises, clicks, blows, hisses or rubs.

The difficulty in describing quality of sound in words is well known. Who can describe in words the difference of the tones of a violin, a flute and a clarinet? Hence many terms have been used for the qualities of heart murmurs. It would be a definite advance in the description of physical signs in the heart if the unnecessary syno-

nyms could be abandoned and if one set of adjectives could be adopted. This would be almost too much to expect at present (Table VI).

TABLE VI

TABLE OF TERMS TO BE USED IN DESCRIBING HEART-SOUNDS AND MURMURS *

HEART-SOUNDS			
Intensity	Quality	Duration	Time
Normal	Normal	Normal	
Faint	Sharp	Short	
Weak	Snapping		
Distant	Valvular		
Muffled			
Loud	Booming	Prolonged	
Accentuated	Muscular		
Increased			
Absent	Split		
Replaced by a murmur			
	Reduplicated		
	Ringing		
	Clanging		
MURMURS			
Faint	Blowing	Short	Systolic
Soft	Soft		
Moderate	Harsh	Moderate	Early systolic
	Rough		
	Coarse		
Loud	Musical	Long	Late systolic
	Rumbling		Diastolic
	Crescendo		Early diastolic
	Decrescendo		Mid-diastolic
			Presystolic (Late diastolic)

* From *Criteria for the Classification and Diagnosis of Heart Disease*, American Heart Association, New York, 1928.

Site.—The site of the murmur gives us much of the information needed to tell the origin of the sound and so to diagnose the lesion. But the study of the site must be complete. Beginners, and many old enough to know better, are too often content to listen at the three classical places, apex, pulmonic and aortic areas, and to stop there. The mistakes made by this plan are many. In the first place, the maximum site may be at none of these areas and, indeed, the murmur

may not be audible at any of them, and so may be completely missed. Again, a murmur heard over the entire precordium is often described as two, one heard at apex and one at the base. This seems almost incredible, yet it is a fairly common mistake made by students and younger physicians. A proper study of the site of a murmur should first determine the *total area* over which it is audible. In other words, when a murmur is heard over the heart it should be traced in every direction until it is lost. In this way the area of audibility is mapped out on the chest wall and should be noted carefully or marked upon the skin. (A skin pencil may be used, but is not always at hand. A fountain pen does very well, the back of the pen being used and held flat against the skin.) The importance of mapping the total area cannot be overemphasized because it insures a complete study of the whole anterior and even posterior chest.

In doing this the *maximum site* is also determined automatically and since this tells us much of the site of production of the murmur it should be accurately ascertained. The man who listens only at the three classical areas will miss many murmurs entirely. He who fails to map the total area will often miss the maximum site which is very frequently at some quite unexpected place, to the right of the sternum or high up in the first right or left space (Fig. 22).

The *direction of maximum transmission* is also observed in studying the total area. This has a bearing on the diagnosis. For instance, a loud rough murmur maximum inside the apex which is transmitted

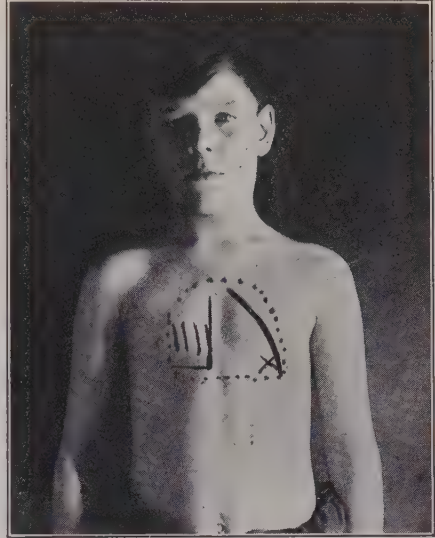


FIG. 22.—GRAPHIC REPRESENTATION OF PHYSICAL SIGNS.

Heart (F. G., aged 9 years). No symptoms.
Apex: Fifth space, 9 cm. from midline.
Left border: 9.5 cm. from midline.
Right border: 2.5 cm. from midline.
Action: Slow, regular, forcible.
Sounds: Normal. Third heart-sound fourth space.
Murmur: Systolic, long, hissing, high-pitched, moderately loud. Maximum to right of sternum. Heard over whole precordium as soft, low blow. Not affected by posture, exercise or respiration.
Diagnosis: Congenital septal defect.
 Class. 1. (A careless observer called this mitral disease.)

loudly to the left beyond the apex is probably mitral in origin. A similar murmur loudest in the same location, which is transmitted more toward the sternum, or beyond it, and not heard outside the apex, is more likely a septal defect.

The quality varies over different areas. At the maximum site and over the area of loudest transmission, murmurs are often rough or harsh, yet are heard as a softer blow over a considerably larger area.

Effect of Posture.—The effect of change of posture on murmurs should always be tried. It is surprising how many are changed considerably by this procedure. It has long been noted that the murmur of mitral stenosis changes when the patient lies down and often appears only in the horizontal position, or when lying on the left side. Other murmurs vary somewhat with change of position, especially the functional or cardiorespiratory varieties.

Respiration.—Respiration also affects heart murmurs and each patient old enough to obey commands should be made to take a long, slow breath while the examiner is listening. Cardiorespiratory murmurs change, as a rule, sometimes becoming louder, sometimes disappearing on deep inspiration. Other murmurs may be lessened in intensity by the overlapping of the lung.

Exercise.—Exercise is useful at times in bringing out some of the qualities of murmurs and even in inducing a murmur. For instance, a short mid-diastolic sound may resemble a third heart-sound, but after exercise becomes a presystolic rumble which shows the presence of mitral stenosis. In another group this sound disappears after exercise and so must be diagnosed as a third sound. Exercise sometimes makes a murmur louder and easier to study, though the increase of the heart rate often makes the matter more difficult. Cases of well-compensated mitral lesions may have no demonstrable murmurs when at rest and in the upright position. If the patient is made to exercise and is placed in the recumbent position on the left side, a presystolic rumble is not infrequently produced. No heart has been thoroughly examined until this procedure has been performed. It is inexcusable, particularly when a patient has a history of rheumatic fever, not to use every possible means in making a diagnosis.

Exercise, by increasing the heart rate, often stops an arrhythmia, such as premature systoles, or it may induce premature contractions. Its effect on the heart action, force and rate is of importance in judging the function and probable integrity of the heart muscle.

Thrills.—The presence of a thrill should be tested, although its importance is often exaggerated. Thrills are best detected by laying the fingers or the whole hand flat on the chest with moderate pressure. If the pressure is too light very slight thrills are palpable in many cases, even in normal hearts. This can be explained by the vibration set up by the contracting muscle as it impinges against the comparatively thin chest wall of a child.

The most common thrill is presystolic, found in mitral stenosis, and here it has real significance because it is felt only when definite narrowing of the mitral orifice is present. The systolic thrill of aortic stenosis in the first and second right space is also an important sign. Simple mitral regurgitation may give a thrill; this is not realized by many who think that only mitral stenosis produces this physical sign. Even aortic regurgitation has been noted by us to be associated with a distinct thrill. Congenital lesions often give thrills. In pulmonic stenosis this is found in the second and first left space. Patent ductus arteriosus less commonly gives a thrill, and septal defects still less often.

Pulse.—The pulse must not be forgotten in the study of the heart. In the infant the artery is small and difficult to feel. For some reason the infant often objects more to having his pulse taken than to any other part of the examination, probably because he dislikes the feeling of being held. The force of the pulse is not easy to judge in infants and small children because of the small size of the artery and the small excursion of the pulse.

The pulse of older children is much like that of adults, but the arterial walls are rarely thickened except in advanced nephritis. The size of the artery, force of the pulse wave, the rapidity of its rise and fall, dicrotism and excursion should be estimated and particularly whether each pulse wave has the same quality and force of every other. The force is usually less than in adults.

In aortic regurgitation there is not often, in childhood, a typical Corrigan pulse. The unthinking observers may rule out aortic disease because of the absence of a Corrigan pulse, but it must be remembered that the valve lesion is recently developed, that the regurgitation is small in amount, that the ventricle is not much hypertrophied and that the arteries have not had time to enlarge because the lesion has only existed a short time. It is occasionally surprising, however, to find a very large pulse pressure with the diastolic pressure almost at zero, with a pulse which, while soft, does not suggest the Corrigan pulse of adults.

The small excursion of the pulse of aortic stenosis is quite characteristic since so little blood is forced into the arteries at each systole. The pulse fills slowly and falls slowly, in contrast to the Corrigan pulse of aortic insufficiency.

Blood-Pressure.—Blood-pressure readings in children with rheumatic heart disease uncomplicated by nephritis show little variation from the normal, except in the presence of aortic insufficiency. In determining the blood-pressure, a mercury manometer should be used, with a child's size cuff, and the reading taken by the auscultatory method, rather than by palpating the radial pulse. The test should be made with the child perfectly quiet; if he has had a meal within an hour or been active physically within fifteen or twenty minutes, he should rest before the reading is taken. Excitement or emotional disturbance of any kind affects the blood-pressure. A number of studies have been made to show the average blood-pressure at different ages in children. The table given is taken from Lincoln's clinical study of 325 children in a private school in New York. It will be seen that the pressure increases with age, that there is no great sex difference, and that the difference between systolic and diastolic pressures lies between 27 and 31, except in girls of three years where the pulse pressure is 22. Tables such as these can serve as a rough guide to the normal blood-pressure. It must be pointed out that there is considerable individual variation and that some persons have normally a higher pressure than others (Table VII).

TABLE VII
AVERAGE MEASUREMENTS OF SYSTOLIC AND DIASTOLIC BLOOD-PRESSURE*

AGE	BOYS		GIRLS	
	Systolic	Diastolic	Systolic	Diastolic
3	93	66	89	67
4	94	67	93	66
5	98	70	95	67
6	98	70	97	69
7	98	70	99	70
8	100	73	100	71
9	101	72	101	71
10	105	75	104	74
11	104	73	105	74
12	102	73	104	75
13	105	75	105	74

* Modified from Edith M. Lincoln, "The Hearts of Normal Children," *Am. J. Dis. Child.*, Chicago, 1928.

The blood-pressure in aortic insufficiency has a high pulse pressure due particularly to the drop in the diastolic reading. A boy of ten who should have a systolic reading of around 105, and a diastolic of 75, will be found to have, in the presence of aortic insufficiency, a reading of 100/50 or even lower. Sometimes the beat can be heard down to zero.

METHODS OF RECORDING PHYSICAL SIGNS

In most hospitals and physicians' offices observations made in the physical examination are recorded by a written description of the signs found. For various reasons these written notes are often inaccurate and misleading. Anyone who has reviewed a number of hospital histories will recall many discouraging misstatements and omissions, often of the essential facts. There are certain inherent difficulties in this matter. First is the carelessness of the intern, who has no thought beyond the day's routine, and who is naturally more interested in the patient than in the record. Next, there is the difficulty of putting into clear language even the simplest scientific fact, yet the physical examination elicits many facts of various kinds not easy to describe. It has grown to be the custom to use set phrases for these records, with many condensed terms handed down from generation to generation of interns. Some of the terms used are adequate enough, because they have become crystallized in medical nomenclature and nearly everyone knows what is meant by them. (Thus the words used to describe the different murmurs in general convey the same idea to different minds, *e.g.*, "mitral systolic," "aortic diastolic," etc.) It would be impossible to do without such code terms, and they do well enough for the average case. But the written descriptions of the distribution of the signs found are apt to be far from correct.

Errors frequently creep into the traditional methods. The worst of these is that of using abbreviations. These change from year to year and are forgotten so soon that it may require some exercise of the imagination to decipher them. For example, "P.M.C.I." is all very well if one knows the answer (point of maximum cardiac impulse) but it is not always easy to guess the meaning even from the context. This type of abbreviation should not be used because the record may be made worthless to another medical generation.

It is advisable to write out the signs elicited following some such outline as given in the chapter on methods of examination, but the graphic method indicates some of the signs better and more quickly

than any written description. A simple plan will be described which has proved of distinct value in showing the progress of cardiac cases for the last ten years.

The site of the apex is shown by a cross, the size of the cross showing the area over which the impulse is felt. The borders are marked by a solid line. The action and sounds are better described in

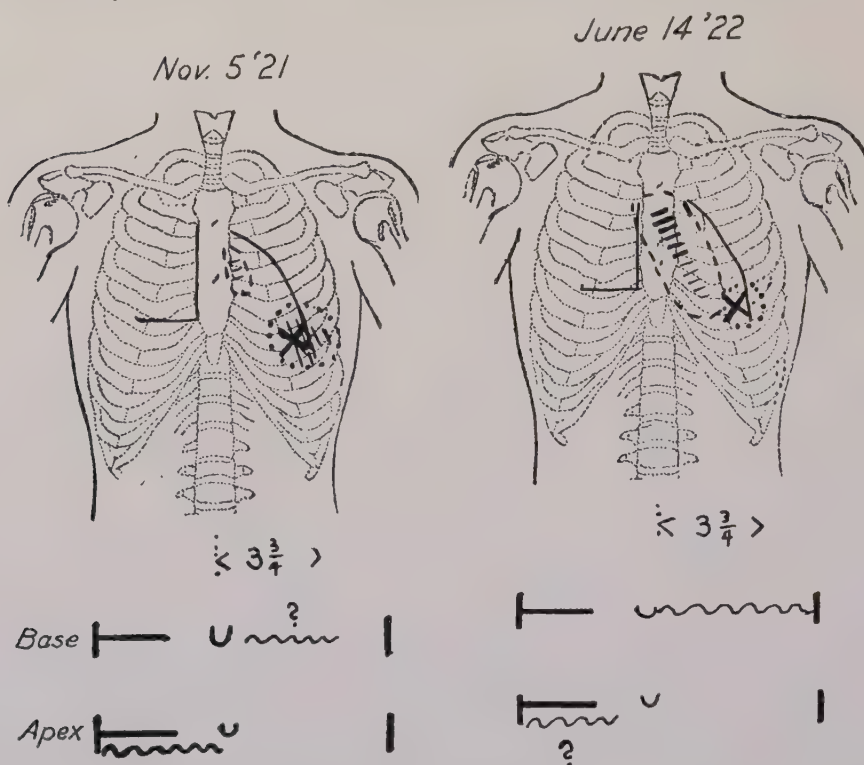


FIG. 23.—PROGRESS OF HEART LESIONS IN A BOY WHO HAD RHEUMATIC FEVER AND CHOREA.

Nov. 5, 1921. There was a definite systolic murmur at apex, a very indistinct diastolic murmur along the left sternal border.

June 14, 1922. The diastolic murmur was louder and more widely heard. The systolic murmur at the apex was very faint.

words than by a diagram. It is possible to show simple arhythmias with a diagram, but the electrocardiogram, of course, does this much better.

It is in recording murmurs that this plan is of greatest value. The qualities of a murmur are divided into three groups in the scheme noted above. The first group which has to do with the qualities of sound can be shown by a code. The cardiac cycle is indicated by a

rough diagram, the first sound by a horizontal line, the second by a curved one. This is mainly useful in indicating the proper relation in time between the murmur and the heart-sounds. The other qualities may be suggested roughly as shown below the diagram in Figures 23 and 24.

But it is in the second group, dealing with the site of the murmur,

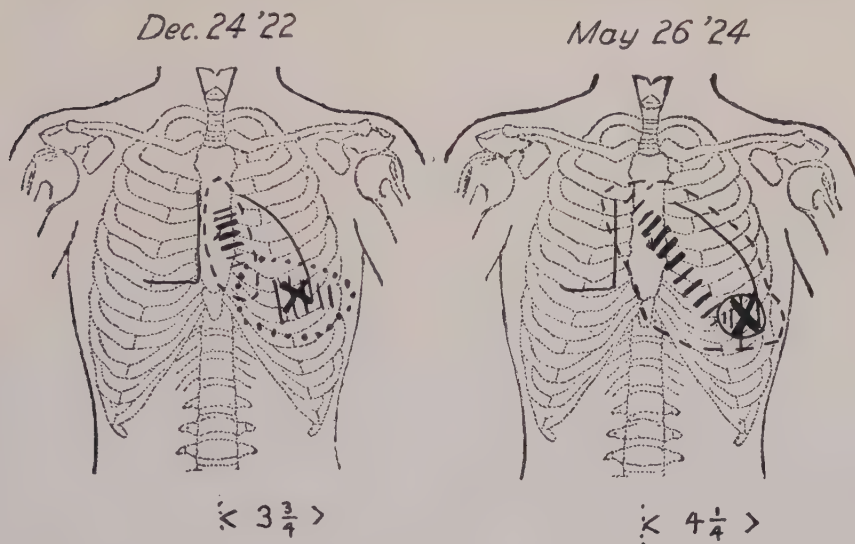


FIG. 24.—PROGRESS OF HEART LESIONS IN A BOY WHO HAD RHEUMATIC FEVER AND CHOREA.

Dec. 24, 1922. The diastolic murmur was definite and the systolic murmur was also heard over a wider area.

May 26, 1924. The heart is much hypertrophied. There is a long, loud diastolic murmur, merging into a presystolic at the apex, but no systolic murmur audible.

A record of this kind shows the progress of cardiac cases better than any possible written description.

that the graphic record has mainly helped us to improve our records. The total area over which the murmur is heard is outlined with *dots* for a systolic murmur, with *dashes* for a diastolic and with a solid line for a presystolic. The maximum site and transmission are shaded in with parallel lines slanting slightly to the right for systolic and to the left for diastolic, with vertical lines for the presystolic. This makes it possible to indicate the maximum of two murmurs at the

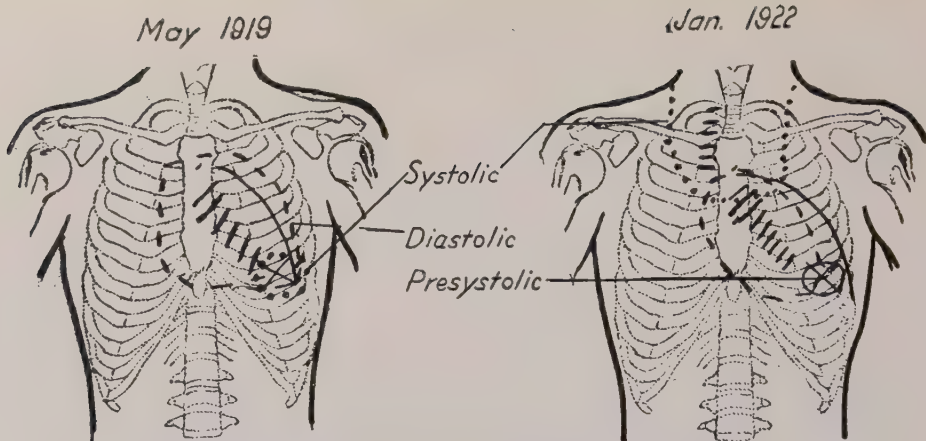


FIG. 25.—GRAPHIC METHOD OF RECORDING OBSERVATIONS.
Change in murmurs over a period of years is clearly shown.

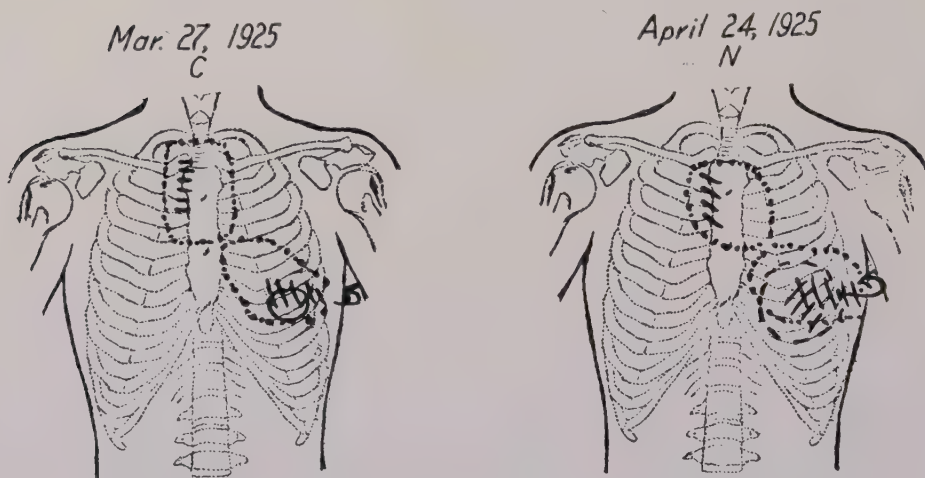


FIG. 26.—IDENTICAL OBSERVATIONS MADE BY DIFFERENT OBSERVERS.

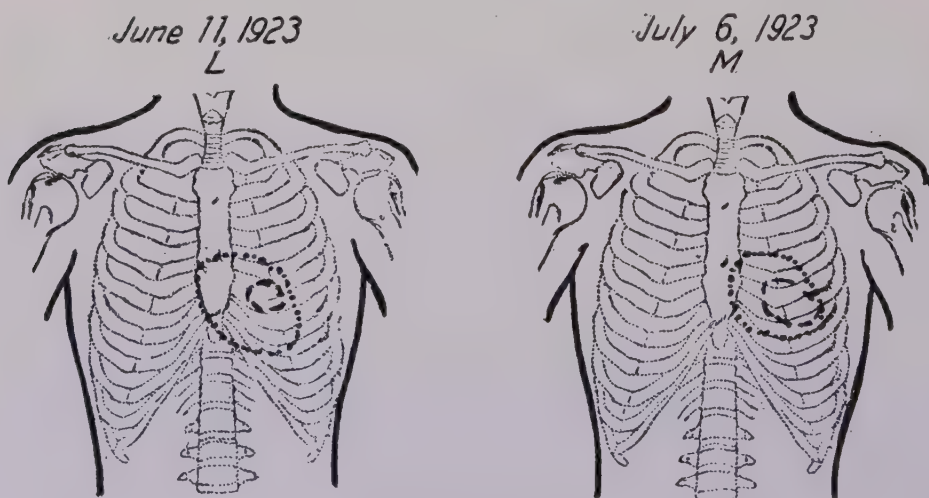


FIG. 27.—IDENTICAL OBSERVATIONS MADE BY DIFFERENT OBSERVERS.

same site by crosshatching, as in a double aortic or a patent ductus arteriosus. Various types of valvular lesions are shown in Figures 25, 26 and 27.

This is far from an exact method but merely a rough clinical one. Its value lies not only in making it quick and easy to record an examination of the heart, but also in stimulating the examiner to greater accuracy in making his record. One is much more careful in noting the exact maximum and total area of a murmur if he has to make a diagram of it, than if it is to be loosely described in words. A careful search for the extreme limit at which the murmur is audible often brings out the surprising fact that it is maximum in some quite unexpected place (to the right of the sternum or high up in the first left space) (Fig. 28). In other words, outlining the total area of audibility necessitates a careful study of the whole chest instead of merely the classical apex, aortic and pulmonary areas, and saves many mistakes in diagnosis.

The progress of a heart case can be recorded from year to year in a more satisfactory manner than by written descriptions (Fig. 29). It often happens that there is marked change in the signs found at one examination from those noted at a previous one. When the former examination was made by a different man there is always a feeling that perhaps the difference in findings may be due to personal equation or carelessness.

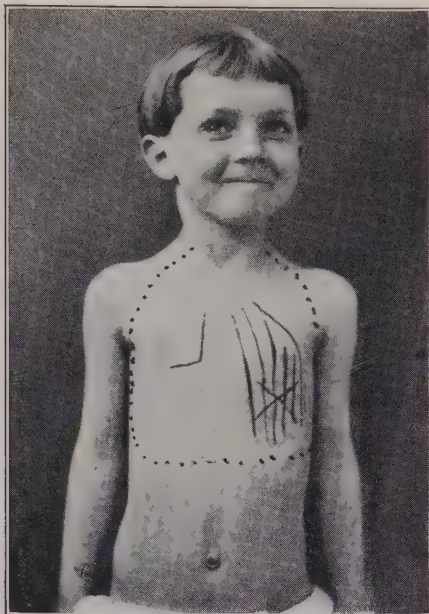


FIG. 28.—HEART LESION FOLLOWING ACUTE RHEUMATIC FEVER.

This child had an attack of acute rheumatic fever at the age of 3 years, with fever, leukocytosis and fugitive joint pains. She had a very loud murmur on admission, which was so loud at the base that it was thought at first to be a congenital murmur. As she has grown older (now nearly 8 years) the murmur is more like a mitral murmur, but is still so loud over the whole chest that there is no real maximum.

Heart

Size: Moderately enlarged.

Apex: Fifth space 5.5 cm. to left, rather diffuse.

Action: Regular.

Sounds: Second pulmonic accentuated.

Murmurs: *Systolic*, rough, loud, long. Heard over entire chest, maximum over whole precordium and outside apex.

Thrill: Slight systolic.

But the diagram leaves no such feeling. If the first examiner has taken the trouble to outline carefully the total area, maximum site and transmission of a murmur, it is very likely to be accepted as an accurate observation. This is not true when the record merely states, for example, that a "double mitral murmur" was present. The tendency to doubt even one's own former observation is very largely removed if a careful diagram was made. The change in signs in heart disease may show an improvement or the reverse. Such changes are shown in Figures 30 to 35 and 36 to 41.

Photography gives us the one means of transferring to paper a real record of the appearance of the patient, which in many instances

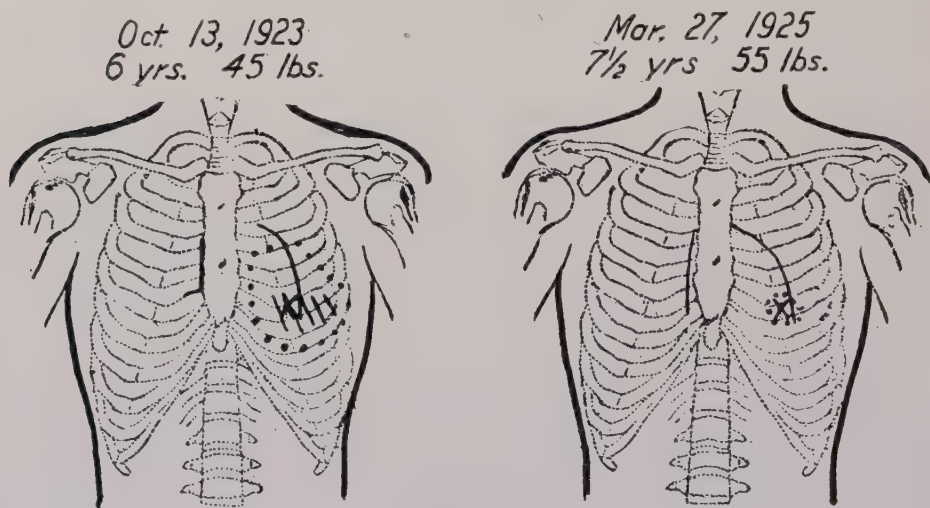


FIG. 29.—DISAPPEARANCE OF MURMUR.

is the most important physical sign in the case. Photographs have long been used in nearly all hospitals and little need be said of their usefulness. An ideal arrangement would be to have one taken of every patient on admission, and repeated later if warranted by any change in aspect. The objection is that photographs are expensive, require a considerable equipment and consume much time. Even if a hospital can afford a full-time photographer, and an elaborate gallery, the time and expense involved would almost prohibit taking photographs of every patient.

It is possible to extend the graphic methods described above by marking on the patient's skin with black water-color paint the physical signs in heart, lungs or abdomen. This gives a record more nearly like

May 1920
S



FIG. 30.

Dec. 1922
McK



FIG. 33.

Jan. 1921
S



FIG. 31.

May 1925
P



FIG. 34.

Nov. 1921
G



FIG. 32.

Oct. 1925
W



FIG. 35.

FIGS. 30-35.—CHANGES IN PHYSICAL SIGNS IN SAME PATIENT AS RECORDED BY VARIOUS OBSERVERS.

July 1921
McK



FIG. 36.

Jan. 1922
M



FIG. 37.

Jan. 1923
M



FIG. 38.

May 1925
R

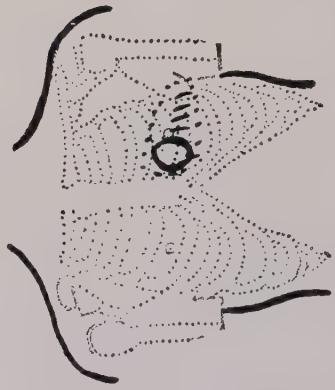


FIG. 40.

July 1925
K



FIG. 41.

Jan. 1924
G



FIG. 39.

FIGS. 36-41.—CHANGES IN PHYSICAL SIGNS IN SAME PATIENT AS RECORDED BY VARIOUS OBSERVERS.

the patient himself than any written words or printed diagram (Fig. 42). These photographs have been of real value in teaching physical signs in heart disease to medical students. A set of lantern slides made from them shows the whole section at the beginning of each session on



FIG. 42.—MITRAL STENOSIS AND INSUFFICIENCY.
Systolic and mid-diastolic murmurs.

heart disease. This saves time by refreshing the memory of the student as to the site and distribution of the murmurs, which are the signs upon which he must mainly rely in making a diagnosis of the valve lesions.

A good photograph of a rare case is a fair substitute for the patient, for, naturally, not all rare conditions can be shown to all students.

CHAPTER VIII

PHYSICAL SIGNS OF HEART DISEASE

MITRAL INSUFFICIENCY

This lesion is the one most frequently seen after acute rheumatic fever and, therefore, should be watched for most carefully during and after this disease. Its onset is often insidious and unsuspected by the patient or the parent because the heart itself may give no symptoms and the diagnosis is frequently missed by the careless medical observer. In this type of case there is no disturbance of the heart action, no enlargement and nothing but the murmur during the early weeks to warrant a suspicion of an acute carditis.

In other cases the lesion is developed more rapidly and the heart muscle is affected sufficiently to give symptoms early as well as the signs which go with disturbed cardiac function. Some cases go on progressively. The heart is involved soon after the first onset of the rheumatic fever and grows worse with each succeeding week in spite of treatment. In children who have repeated attacks of rheumatic fever the heart is often involved with each, suffering more and more valvular and muscular damage.

The signs of mitral disease vary with the type of case and with the degree of valvular and myocardial involvement.

In the mildest cases the size of the heart as judged by the apex impulse and the border is not appreciably increased. The action is regular, slow and forcible. The sounds are clear. The first sound has a normal quality and is not replaced by the murmur. The pulmonic second sound is not accentuated, though it may be louder than the aortic as is normal in any child. The murmur is a systolic blow heard after the first sound and filling all or part of the interval between first and second sound. Its quality varies from a soft blow to a slightly rougher hissing sound. The intensity also varies and may be very faint at first, but later the murmur becomes moderately loud with the development of hypertrophy of the ventricles. The maximum site is usually at or near the apex and the maximum transmission is to the left (Fig. 43). These murmurs are practically always audible outside the apex, even

when not very loud. The total area over which the murmur is heard varies greatly. The faintest murmur may be heard only over a small area around the apex. Louder ones can be heard over more of the precordium and more to the left. In many cases the murmur is audible as far upward as the pulmonic area and may be mistaken here for a different murmur. If the examiner takes care to map out the total area over which it is heard, it is evident that only one murmur is present.

The pulse is normal or may show second or third degree heart-block.

The efficiency of the heart and its response to exercise is not appreciably affected.

To summarize, the murmur is practically the only physical sign. It may not be possible to differentiate this murmur from the soft systolic apical murmurs which we know as functional or "possible" murmurs. Only time and repeated careful observations will make the diagnosis. Such murmurs may disappear and reappear later. Mitral insufficiency should not be diagnosed on the murmur alone but only in the presence of a murmur plus enlargement of the heart. The enlargement may not be demonstrable clinically, but may be shown clearly in the 2-meter x-ray plate.



FIG. 43.—MITRAL INSUFFICIENCY.

Outline of heart and of murmur marked on chest.

In most advanced cases the size of the heart is definitely increased. The maximum impulse is displaced to the left of the normal site for the age of the child. The left limit of the palpable impulse may be one-half inch or more to the left of the normal position (well outside the nipple line). The character of the apex impulse suggests slight hypertrophy, *i.e.*, it is somewhat more diffuse than normal, being felt over an area $\frac{1}{2}$ to 1 inch in diameter. The impulse has a more forcible thrust than normal.

The heart-sounds are altered. The first sound is more booming,

the second pulmonic is distinctly accentuated. In the more advanced cases in this group the first sound may be partly or entirely replaced by the murmur. The heart action at rest may be but little affected, slow and forcible, but the rate increases on exertion somewhat more than in a normal child. The murmur is usually loud and often harsh and hissing, occasionally musical. There is sometimes heard a shrill, whining, high-pitched sound which simulates the cry of the seagull. The murmur occupies practically all of the systole, even when the first sound is not replaced by it. Its maximum transmission is toward the axilla and even to the back. The murmur is heard over all of the precordium and often beyond it. Frequently the hissing or harsh quality is lost at a distance from the maximum site and area of maximum transmission and the murmur is merely a softer blow over the rest of its area of audibility.

There may be a systolic thrill at the apex when the insufficiency of the valve is marked. The pulse is usually normal or slightly full. The blood-pressure is not regularly affected.

The cardiac efficiency is not impaired at rest, but the exercise tolerance may be decreased. The extent of the disability varies. Many of these children are perfectly comfortable at rest and do not complain of dyspnea or distress on exertion, but the heart rate increases unduly and they are forced to breathe more rapidly and deeply after running or stair-climbing. They have no edema. Most of this group are able to go to school and to lead fairly normal lives if they can be taught their limitations and if they will learn what they can do and what they cannot do. The uncoöperative child does less well. The severer cases can walk fairly well, but soon find for themselves that they cannot run. They have to take stairs slowly or rest between flights or pay for their overexertion with a period of uncomfortable overaction and dyspnea.

MITRAL STENOSIS

Mitral stenosis is a lesion diagnosed in children more often than is warranted by the physical signs present. It is true that at autopsy nearly every child who has died of heart disease has some narrowing of the mitral orifice or stiffening of the valve. But it must be remembered that children who die of heart disease have had progressive valvulitis which has existed for a comparatively long time, during which the stenosis has taken place. The mild ambulant case with small involvement does not necessarily have stenosis, even with definite ev-

idence of insufficiency. Stenosis does not often occur in young children but is usually seen in older ones who have had repeated attacks of rheumatic carditis. It is not justifiable to make this diagnosis without some evidence, except in older children with long-continued disease. In these the real signs are often present.

The size of the heart is less affected by pure mitral stenosis than by any other lesion, because the extra work is thrown on the auricle rather than on the ventricle. The auricle may hypertrophy but this is not easily recognizable. The x-ray may show an exaggeration of the left auricular curve.

The heart action in the milder cases is not bad, although it is easily disturbed by exercise. The first sound is characteristic, short, loud, snappy, valvular in quality. The second pulmonic is markedly accentuated.

The typical murmur of mitral stenosis is late diastolic, ordinarily called presystolic. It is rumbling in quality and runs with a crescendo up to the snappy first sound. The maximum site is usually just inside the apex, but it may be audible over an area extending from the sternum to the apex. It is rarely audible up to the base of the heart. It has no especial direction of transmission (Fig. 44).

This murmur is practically indistinguishable from the presystolic rumble heard in aortic regurgitation (*q.v.*), and mitral stenosis should never be positively diagnosed on a presystolic murmur in the presence of that lesion.

The murmur of mitral stenosis is extremely variable. It may come and go or may change its character from hour to hour. Not infrequently it moves from a presystolic to a mid-diastolic murmur, loses its crescendo character, becomes short, almost valvular, and may easily

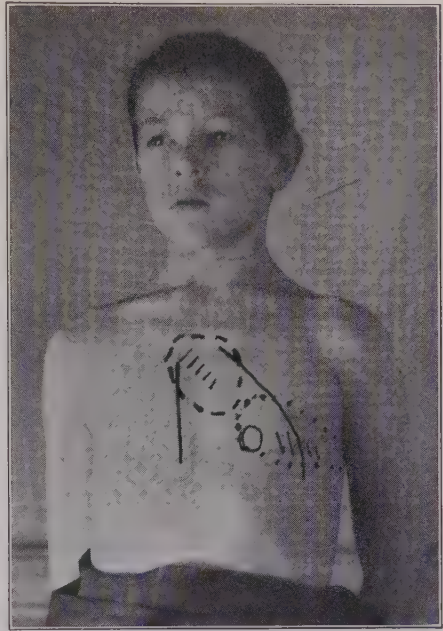


FIG. 44.—MITRAL STENOSIS AND INSUFFICIENCY, AND AORTIC INSUFFICIENCY.

Systolic and presystolic murmurs outlined over mitral area. Diastolic murmur outlined over aortic area.

be mistaken for a third heart-sound. Students usually call this a duplicated second sound, but it is only heard inside the apex over a limited area and there is no splitting of the second sounds at the base of the heart. From a third heart-sound it can be differentiated by exercise in many cases. Increasing the heart rate frequently causes the short mid-diastolic murmur to move up into a typical presystolic rumble; when the heart slows again it may return to a mid- or even early diastolic murmur.

The third heart-sound usually disappears with the shortened diastole of a rapid heart rate. Exercise may bring out a presystolic murmur in cases which are perfectly normal on examination at rest. Such a case was that of a boy of twelve years who had had several rheumatic attacks and had, for a long time, a mild mitral systolic and later a short presystolic sound. As he grew older and developed physically, at puberty both murmurs apparently disappeared. But, knowing that they had been present, he was made to exercise, when a definite presystolic rumble appeared for a very short time, becoming inaudible as soon as his heart slowed down.

Change in posture brings out the murmur of this lesion not infrequently, even though it is quite absent in the erect position. It is therefore extremely important to examine the child lying down and after brisk exercise, before ruling out mitral stenosis.

The more serious lesions are usually associated with regurgitation, but a tight stenosis occasionally occurs alone (Fig. 45). In these, there is little enlargement but the heart action is poor and attacks of congestive failure often supervene. The murmur is loud and rumbling, the first sound very short, the second pulmonic very loud.

Double mitral lesions are more common than simple mitral stenosis in the first decade of life. The insufficiency is the first lesion to appear during an attack of rheumatic fever, at first relative insufficiency, then with increasing valvular damage a true valvular incompetence as well. Then narrowing of the orifice takes place by adhesion and deformity of the flaps and stenosis is added. Finally, the funnel-shaped valve orifice develops if the patient lives long enough, and the process continues. The involvement of the heart muscle going on at the same time leads to dilatation and then hypertrophy, so that these hearts are enlarged, weakened and handicapped by narrow and incompetent valves. They often go from bad to worse and fail to carry on the circulation.

The signs of mitral insufficiency and stenosis are those of the two lesions combined, often with one or the other predominating.

The size of the heart is large, often extending to midaxilla. The apex is usually diffuse and heaving, but with little force to its thrust.

The action varies with the severity of the case; it may be regular and forcible in the milder ones, but is apt to be of poor force and quality in the more advanced lesions.

The first sound may be present, and of normal, or snapping, quality, or may disappear entirely, being replaced by the systolic murmur. The second pulmonic is definitely accentuated, except with a failing

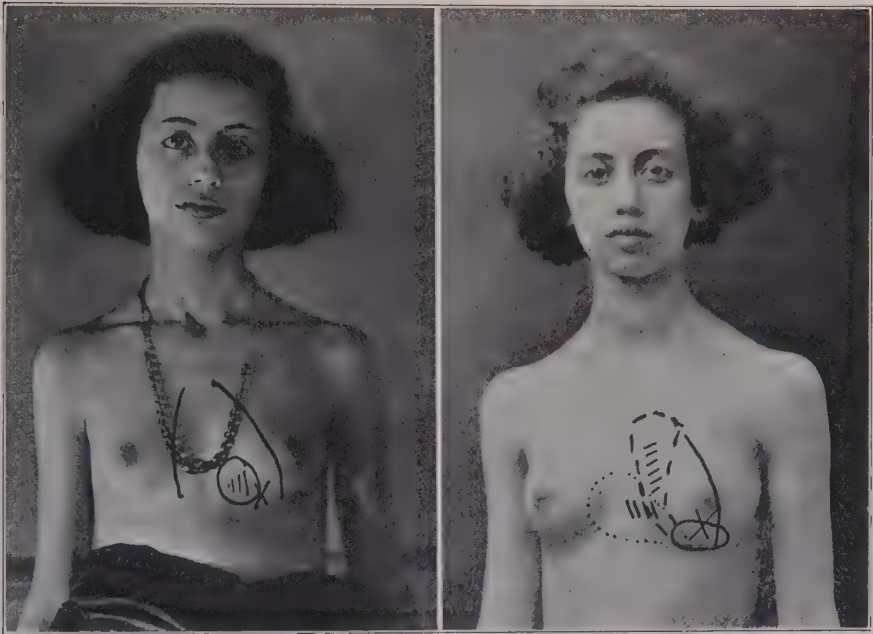


FIG. 45.—MITRAL STENOSIS FOLLOWING CHOREA.

FIG. 46.—SAME PATIENT AS IN FIGURE 45.
Aortic insufficiency has appeared one year later.

right heart. The mitral systolic murmur varies greatly in loudness and quality, but is usually fairly loud and rough or musical.

Double mitral sound is present except when the heart fails, in which case it may be almost or quite absent. Its site and distribution vary as in simple mitral regurgitation.

The presystolic murmur is just as variable as when heard alone. It may be a good presystolic rumble at all times, but often disappears or moves into mid-diastole. The combination of systolic murmur, second sound, mid-diastolic sound at the apex or just inside it, gives the so-

called "who took it" sequence, commonly known as "gallop rhythm." It should be called "gallop sound," or simply "gallop," for there is no alteration in rhythm. This gallop is heard regularly in severe mitral disease and usually means serious myocardial involvement. It may be impossible to tell whether the mid-diastole sound means a mitral stenosis or a third heart-sound, because these patients are often unfit for any exercise test.

AORTIC INSUFFICIENCY

This lesion usually comes on later than the mitral involvement, although it may be the first to appear. In the Huddleston Cardiac Class at Bellevue Hospital, the average age of aortic cases when the aortic lesion is first discovered, is definitely older than that of mitral cases, about ten years, but occurs in many as early as six years of age.

Aortic insufficiency is syphilitic in origin in many adults, but it is often part of the rheumatic heart disease in children so that a large number of adult cases of aortic disease must date back to childhood.

The first sign is the murmur with few or no other findings.

In the mild cases the *size* and *action* of the heart are not appreciably affected. The first heart-sound may be slightly more muscular, while the second aortic sound is weaker or absent. The typical murmur is a faint, soft, low-pitched blow, heard throughout diastole. Its site is along the left sternal border from the third to fifth spaces. It is often heard through the sternum but may not be audible at the aortic area (second right space) in the early stages (Fig. 46). The total area is often not large, perhaps $\frac{1}{2}$ by 1 inch. These murmurs are frequently missed entirely and are, therefore, considered to be uncommon. The type of stethoscope used is most important. In our experience the shallow parabolic bell picks these murmurs up when they cannot be heard at all with the deep funnel or cone-shaped bell. Students regularly miss these murmurs unless they have good stethoscopes.

When the murmur is louder, it is usually heard easily and over a wide area, extending upward to the second right space, downward to the apex or even beyond it. The total area is usually oval with the long axis obliquely placed from aortic area to apex. The second aortic sound is replaced by the murmur. The second pulmonic is generally accentuated.

The signs associated with a marked aortic leak are not present in the early cases, that is, Corrigan pulse, pistol shot in the peripheral vessels, capillary pulse, and so forth. Their absence often makes the

tyro question the diagnosis. It must be evident that these signs depend upon a large leakage of the valves which, at first, is not present. A small erosion or distortion of a semilunar cusp may be quite enough to allow leakage and give a murmur, but not enough alteration in the circulation takes place at first to give the signs of advanced disease.

When the regurgitation has lasted for a longer time and when more blood flows back into the ventricle during each diastole, distention of the ventricle takes place, followed in a few months by marked hypertrophy. We then find the classic symptoms of aortic regurgitation.

The size of the left ventricle increases tremendously and the maximum apex impulse is found in the fifth or sixth spaces, well toward the anterior axillary line. The impulse is diffuse and heaving, often palpable in two or three spaces over an area 1 or even 2 inches in diameter. It is palpable to the left often to $4\frac{1}{2}$ or 5 or more inches from the midline. The left border of the heart is well to the left, often in the midaxilla. The right border does not often percuss to the right of the sternum. The x-ray shows a large heart with the left ventricle much hypertrophied.

The heart action is usually good. The hypertrophied muscle contracts forcibly, regularly, and not very rapidly. As a rule, these children have very good compensation when the aortic is the only valve involved.

The first sound is loud and booming. The second aortic sound is entirely replaced by the murmur and the second pulmonic sound is moderately accentuated. The murmur is long, filling diastole, fairly loud, soft-blowing in quality, low-pitched, rarely rough or musical. It is heard most loudly in the second right and third left spaces, then fades off toward the apex. The total area of audibility is seldom as great as that of a loud mitral murmur.

As the diastolic murmur is traced toward the apex it not uncommonly acquires a late diastolic crescendo which is rumbling and resembles very closely the presystolic murmur of a mitral stenosis. This murmur was described by Flint and bears his name. The mechanism of its formation is not quite clear.

It may be that the presystolic rumble of a Flint murmur is not quite so rough and that the first sound is not quite as snappy as in true mitral stenosis, but when there is in addition a mitral systolic murmur it is impossible to differentiate them. It should always be remembered that in the presence of an aortic regurgitation it is not

justifiable to diagnose mitral stenosis merely because of the presence of a presystolic murmur.

All of the secondary signs gradually appear in children with advanced aortic regurgitation, but since the lesion occurs in older children and since it takes some time for it to develop the children who present them are, as a rule, approaching puberty.

The pulse, it is true, becomes gaseous, is not sustained fairly early, but does not become bounding and of the Corrigan type, until the arteries have grown large. In the younger children (eight to nine years of age) with this lesion, it is often impossible to feel the pulse when the arm is held straight up above the head. The pulse pressure is large, the systolic blood-pressure is usually 90 to 110, but the diastolic blood-pressure is very low, often down even to zero.

The pistol-shot sound is easily heard in the large arteries of the larger children. Capillary pulse may also be present. There is rarely edema or other evidence of circulatory failure in simple aortic insufficiency as seen during childhood.

AORTIC STENOSIS

Aortic stenosis is one of the rarest lesions in childhood. Since this condition is secondary, taking place only after the valves have been damaged for a long time, the individual is usually well before stenosis develops. We have seen only three or four definite cases in twelve years (all in boys of eleven to fifteen years of age).

The heart is very large in these children, the apex is in the sixth or seventh space, the left limit well into the axilla. It has a characteristic, slow, heaving, thrusting impulse, which is seen in no other condition. The action is usually slow, very forcible and regular. The first sound is extremely loud and booming. The second aortic sound is usually feeble or absent. The murmur of stenosis is systolic in time, long, rough or rasping, loudest in the first and second right spaces and is transmitted loudly upward, even into the neck (Fig. 47). The total area varies, but is symmetrical about the aortic area. There is, of course, the diastolic murmur of the regurgitation which is usually present, but pure stenosis does occur. There is nearly always a rough thrill over the right upper chest. The pulse is small, has little excursion, comes up and falls away slowly, and in no way corresponds to the enormous thrust of the ventricle. The blood-pressure is not high.

The diagnosis of true aortic stenosis rests on these cardinal signs, the enormous hypertrophy and forcible apical thrust, the rough sys-

toxic murmur at the base, the small pulse, and the systolic thrill in the aortic area at the right of the sternum.

Often, there is a double aortic murmur without the other signs of aortic stenosis. The murmur is usually less rasping than that of stenosis, but may be rough. Its site and distribution are the same as those of stenosis. The heart is hypertrophied, but resembles that of a simple regurgitation; the apex may be forcible, but lacks the extreme thrust seen with stenosis. The pulse is soft or fairly large and bounding in older children, showing that the regurgitation is the predominant factor.

Symptoms in these cases were formerly called aortic regurgitation and roughening. Whether the systolic murmur is formed by the blood rushing through an orifice roughened and distorted by the productive changes in the valves, or whether there is some narrowing in these cases, is often difficult to decide. Rheumatic aortitis may be the explanation.

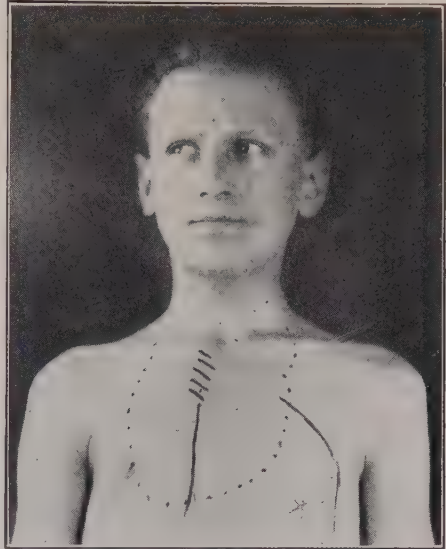


FIG. 47.—AORTIC STENOSIS.

Systolic murmur outlined over aortic area and transmitted up into neck.

TRICUSPID INSUFFICIENCY

This rarely occurs alone as a true endocarditis, but usually is a relative leak due to failing myocardium. Vegetations do occur on this valve, however, and the tricuspid may be the only valve affected in an attack of rheumatic endocarditis.

The signs of tricuspid regurgitation are not often seen and are frequently missed when present. Since it usually occurs only as an end result associated with mitral disease, the signs of that lesion ordinarily overshadow and mask the tricuspid signs.

The heart is already enlarged by many attacks of carditis, and the increase caused by the right heart dilatation is superimposed on the signs of left ventricle enlargement. Enlargement of the right ventricle increases the heart outline downwards as well as to the right,

but the only recognizable increase is to the right of the sternum. This may be percussed as dulness beyond the right sternal border, but only in the presence of considerable enlargement.

The action is usually poor, growing steadily worse as the congestive failure increases. The sounds are poor or obscured by the murmurs, often from several valves. The second pulmonic sound loses some or all of its accentuation. There is apt to be arrhythmia, often auricular in origin.

The murmur of tricuspid insufficiency is loudest just to the right of the lower part of the sternum, and it is transmitted to a varying distance to the right. As a rule, the mitral murmur so regularly present runs over into the tricuspid, and the lesion can rarely be diagnosed by the murmur. Occasionally the mitral sound fades off as one goes from left to right of the sternum. Without this region between the two sides where the murmur is less loud, one cannot be sure of a tricuspid murmur.

The diagnosis is easily made, however, by the signs existing elsewhere than in the heart. The veins of the neck pulsate and this may extend even to the veins of the arm. True expansile pulsation of the veins must be seen, and not merely the lifting of a distended vein by the pulse from the adjacent artery.

The enlarged, tender and pulsating liver clinches this diagnosis. In examining the liver both hands must be used, one in the right flank and one anteriorly. A truly pulsating liver can be felt pushing the palpating hands apart with each systole.

PERICARDITIS

Pericarditis is one of the most characteristic of the lesions of rheumatic fever and the pericardium is involved to some extent in all children dying of rheumatic heart disease during the first ten years of life. When the child's temperature rises suddenly and he complains of pain about the heart, fibrinous pericarditis should be suspected. There may, or may not, be a definite friction rub at first, but it usually develops, although sometimes not until the fever has dropped and the child feels more comfortable. It is remarkable how little inconvenienced the patient may be even in the presence of a very loud friction-sound.

The friction-sound which is the characteristic sign of masses of fibrin on the pericardial layers is usually first heard along the left border of the sternum. The area over which it is heard increases

gradually, until in some cases it is heard over the whole precordium, even to the left beyond the apex and to the right as far as the axillary line. The sound is distinctly a rub, often to-and-fro in character. It does not always synchronize with the heartbeat and may be inconstant. Pressure on the skin with the stethoscope may make the rub appear louder. The fibrin interferes with the smooth interplay of the pericardial layers on each other and causes the actual rubbing-sound. The rub may persist for days or even weeks.

Presence of pericarditis indicates serious infection and there is always a myocarditis at the same time. Symptoms, such as dyspnea, are to be explained by the myocardial involvement, rather than by embarrassment of the heart action from the pericarditis. Skin hyperesthesia over the precordium is not infrequent.

The end result of repeated attacks of fibrinous pericarditis is the formation of adhesions between the two layers of pericardium or between the pleura and the pericardium. This does not often occur at the stage of heart disease which we see in children. When adhesions do form, they constitute a serious handicap to the heart, since the movements of the heart are mechanically interfered with by the adhesions. Fixation of the apex-beat may occur, so that, when the child is put in different positions, the apex remains in the same place. Visible retractions of the chest wall over the apex are occasionally seen. This must not be confused with the bulging of the chest wall, which can be seen when a thin child has a hypertrophied overacting heart.

PERICARDIAL EFFUSION

Pericardial effusion is a fairly common complication of rheumatic carditis. If the effusion is small, there may be no very definite signs and the presence of fluid may be suggested only by the contour of the heart shown in the x-ray. Contrary to the view formerly held, pericardial friction-sound frequently persists even after the development of effusion. Conner states that in twenty-four of thirty-four cases of pericarditis with frank signs of effusion (70 per cent) the friction-sound was noted during the time when the presence of effusion could be clearly demonstrated.

In most cases of large effusion the apex-beat cannot be felt and the heart-sounds become muffled. These findings are not constant, however, and absence of these signs does not rule out fluid in the pericardium, since the heart remains close to the anterior chest wall even in the presence of large amounts of pericardial fluid. The impulse

which is often felt in the third left intercostal space is not the displaced apex-beat, as previously thought. It is the impulse of the left side of the body of the heart, which is felt more distinctly because of the pushing away by the enlarged heart of the border of the left lung. In other words, the position of the heart does not change materially when there is pericardial effusion. Conner states that in pericardial effusion the "most significant early changes from the normal outline of cardiac dulness are the appearance of marked dulness behind the manubrium and in the first and second right and left intercostal spaces; the extension of the absolute dulness of the heart into the right, fifth intercostal space (Rotch's sign), and especially the extension of this dulness to the left of and below the position of the apex-beat. This lower border of dulness in the apical region can usually be readily determined because of its contrast with the tympany of Traube's space." As the amount of fluid increases, the area of flatness assumes a roughly triangular shape, with the right side roughly parallel to the right border of the sternum and a few centimeters to the right of it. The left border in very large effusions extends well into the axilla and merges with posterior flatness. The dulness of cardiohepatic angle becomes rounded instead of a right angle.

PULMONARY SIGNS

A considerable number of children suffering from rheumatic fever develop pulmonary symptoms and signs.

The most common of these conditions affecting the lungs is chronic congestion. This results first in a little cough and slight expectoration. A tendency to bronchitis develops and the symptoms are increased with every cold.

The signs vary from a few râles at the bases to signs of a general bronchitis. When the congestion goes on to the condition of brown induration, signs of consolidation (dulness, bronchial breathing and voice) often appear at the bases. Not uncommonly, true pneumonia complicates rheumatic carditis. This may be an intercurrent lobar pneumonia or a real rheumatic pneumonia. These children have fever which runs high or irregularly, definite signs of consolidation and an x-ray shadow corresponding. We have seen several definite lobar pneumonias complicating carditis, with a crisis and recovery. In some of these, the child who has had orthopnea from the cardiac condition is able to lie down during the pneumonia in relative comfort only to have to sit up to breathe after the crisis. This may be because the

greatly accelerated respirations during the pneumonia better oxygenate the blood. The rheumatic pneumonias are apt to be much more persistent and run a long course. We have seen children with fever and signs in the lungs for one or more months who recovered.

Infarcts occur in acute carditis with varying frequency. They probably come from emboli breaking loose from the thrombi formed in the auricle, and developed in long-standing cases. These emboli may land in the lung, obstruct a vessel and cause ordinary infarcts. The signs and symptoms of these are not unlike a pneumonia and the course may resemble the same disease.

It is not always possible to differentiate infarcts from pneumonia clinically. Some observers think infarction is rare, while others believe that nearly all apparent pneumonias in heart disease are due to this cause. This latter view does not appear to be supported by autopsy findings.

Since it has been demonstrated (as stated in the chapter on pathology) that characteristic changes occur in the lungs in rheumatic fever, it seems reasonable to interpret some of the pulmonary signs as caused by a rheumatic pneumonitis.

The most common site of signs of consolidation is at the left base behind the heart. It has long been the custom to attribute these signs to "pressure on the lung by the heart." But the largest hearts often fail to give these signs, yet there must be as marked pressure on the lungs as in the cases with signs.

It has long seemed to us that there must be lung pathology to account for the signs of consolidation. Whether it is pneumonia, infarction, brown induration, or pericardial effusion, can only be determined by careful study.

In one such case a girl had classical signs of consolidation over the lower third of the left base for weeks before death. Autopsy showed merely a chronic brown induration of the lung.

Another girl of ten years was admitted with a mitral endocarditis, cough, fever and marked signs at the left base. She had cough and profuse expectoration dating back to a severe bronchopneumonia at two years of age. Her left lung showed dulness and feeble respiratory sounds over the lower half, but after coughing up several ounces of purulent sputum she had definite cavernous breathing. Lipiodol showed multiple bronchiectasis. This evidently had nothing to do with her cardiac condition. Yet her signs were attributed by the admitting physician to pressure by the heart.

A boy six years of age, who was admitted to the hospital after one day of high fever and cough, clearly seemed to have a rheumatic pneumonia. He had signs of consolidation at the right base, dulness with pure bronchial breathing, and the diagnosis of lobar pneumonia was made. After two days the temperature dropped and the lung signs disappeared completely without going through any stage of resolution. With the slowing of the heart rate, following the disappearance of fever, he was found to have a definite double mitral lesion with enlarged heart and no pericardial effusion.

The lung signs which occur in cases of pericardial effusion are very interesting and often puzzling. These signs may be marked and occur on both sides. They may be confined to a small area, or there may be no pulmonary signs. Flatness and intense bronchial breathing, or bronchophony over either or both lower lobes, may be present. Usually there are no râles except occasionally crepitant râles along the edge of the flat area. The bronchial breathing has the distinct character often found in pleural effusion. Although these signs occur most often on the left side, they may be found only on the right. The first change is usually a patch of bronchial breathing and dulness between the inferior angle of the left scapula and the vertebræ. This area gradually increases with accumulation of the pericardial fluid until there is flatness over the whole of both or either lower lobe. The inflamed pericardial sac, as Conner points out, can be distended in contrast to the normal pericardium. As the pericardium becomes more and more filled with fluid, the lung retracts, and if the effusion is very large indeed, the lung becomes compressed into a very small area.

Pleural effusion occurs as a complication of rheumatic fever. The signs are no different from those of effusion from any other cause. The only differential diagnosis to be considered depends upon the signs which are found in the presence of pericardial effusion. When there is doubt an x-ray plate will often settle the question.

CHAPTER IX

CONGENITAL MALFORMATIONS OF THE HEART

ANATOMICAL CONSIDERATIONS

Development of the Heart.—It is necessary to review briefly the development of the heart in order to understand congenital malformations. For a more detailed description see His, Hertwig, Kölliker, and others.

The heart is formed from the visceral mesoderm in early embryos (one to two months) as two-paired anlagen just ventral to the foregut. These fuse to form a single tube (or pharyngeal region) of mesenchymal cells lined with endothelium. This tube connects at the head end with the primitive aorta and receives at the caudal end the primitive veins. It soon ceases to be straight, becoming bent into an S-shape by its rapid growth. The arterial end lies in front, the venous end grows up behind. From the latter, two pouches (the atria) grow forward around the arterial trunk and form the auricles. The ventricles thus are formed of a loop with a larger descending arm to the left and an aortic limb to the right. A constriction marks the junction of ventricle and atria (auricular canal) and is the site of the future auriculoventricular valves. The sinus venosus opens into the posterior part of the auricle on the right side, having collected all the venous blood.

The development into a two-sided heart takes place by the formation of three secondary septa:

(a) The auricular septum starts by a crescentic shelf at the top of the auricle which gradually separates the auricles into halves, joining with the septum of the auricular canal (septum intermedium). The foramen ovale is a defect left in this septum. There is a smaller false septum (septum spurium) above the opening of the sinus venosus which later fuses with the true septum and plays a part in the valve formation.

(b) The ventricular septum starts as a crescentic projection at the lowest point of the ventricular loop. It grows upward and joins the septum intermedium to make two ventricles.

(c) The septum intermedium divides the auricular canal into two parts and fuses with the superior and ventricular septa.

(d) The aortic septum is formed by two endothelial buds which grow together and fuse, forming the aorta and pulmonary artery. The spiral direction of this septum causes the twist of aorta and pulmonary artery. It also dips into the ventricle to join the interventricular septum forming the pars membranacea septi.

Classification of Anomalies.—There are several anomalies of the heart, found mostly in monsters of various sorts, which are of merely scientific interest; but have no clinical value since the individuals do not live. Among these may be mentioned: absence of the heart, twins with but one heart, hernia of the heart (ectopy), and so forth.

The congenital lesions which are compatible with life may be classified as follows:

- A. Defects in septal development
 - 1. Interventricular septal defect
 - 2. Interauricular septal defect
- B. Abnormal persistence of opening
 - 1. Patent ductus arteriosus
 - 2. Patent foramen ovale
- C. Deformities of vessel orifices and valves
 - 1. Pulmonic stenosis or atresia
 - 2. Aortic stenosis
 - 3. Mitral or tricuspid stenosis
- D. Abnormalities of vessels
 - 1. Narrowing of aorta
 - 2. Transposition of aorta and pulmonary artery
 - 3. Aorta arising from both ventricles
 - 4. Abnormal arrangement of vessels rising from the bronchial clefts system (subclavian, carotid, etc.)

The most common congenital condition is a combination of two or more of these lesions, usually pulmonary stenosis and septal defects. The most common single lesions are septal defects, pulmonic stenosis and patent ductus arteriosus.

Single Lesions.—**SEPTAL DEFECTS.**—**Interventricular.**—The most common interventricular congenital lesion is a defect in the upper part of the interventricular septum in the pars membranacea, which is

caused by the failure of the aortic septum to join the interventricular septum (Figs. 48 and 49). The defect is high up and varies from a

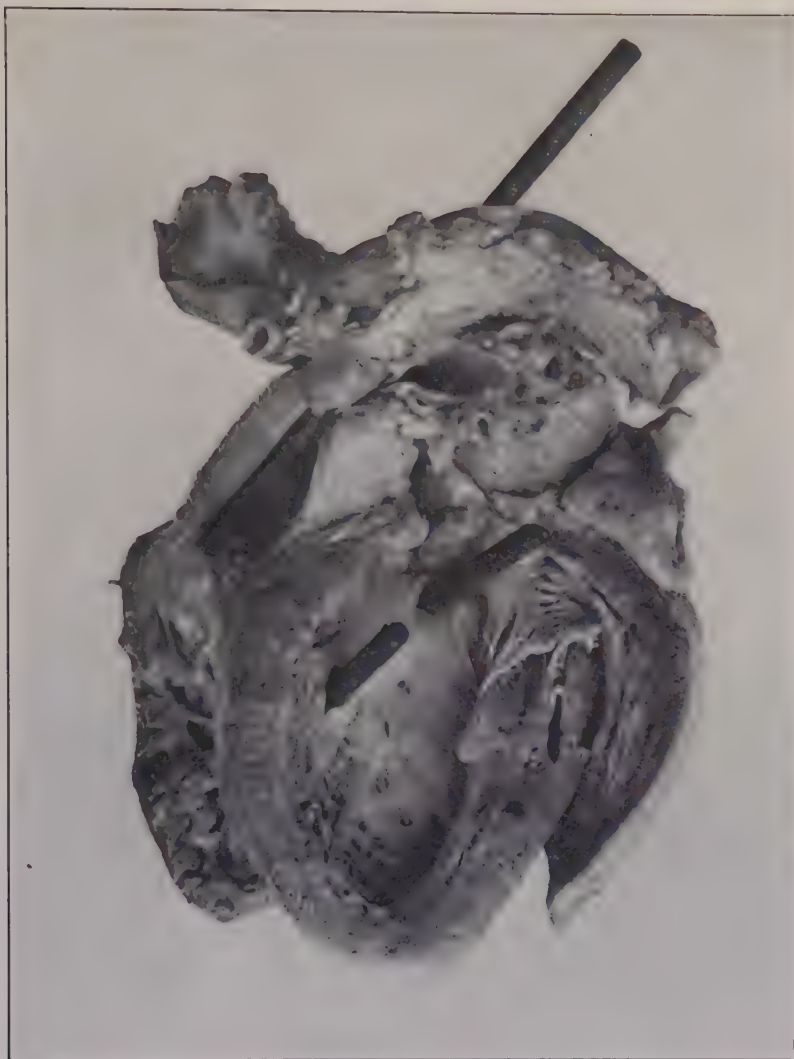


FIG. 48.—TWO DEFECTS IN INTERVENTRICULAR SEPTUM, UPPER OPENING AT USUAL SITE. PATENT DUCTUS ARTERIOSUS.

This infant, 2 weeks old, also had an extra digit and congenital obstruction of the parotid duct. (Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

pinhole to an opening the size of a dime. (The term "patent septum" is a bad one and should be discarded, as patency implies the persistence of a normally present opening, *e.g.*, ductus arteriosus.) The large de-

fects are caused by imperfect growth of the ventricular septum, or even its complete absence, leaving only one ventricle. This is usually fatal in the first few days of life. The smaller defects cause little disturbance in the circulation and may be compatible with perfect health. The opening must be fairly large to cause any cyanosis or functional disturbance.

Interauricular.—A persistent foramen ovale is not rare (Fig. 50)

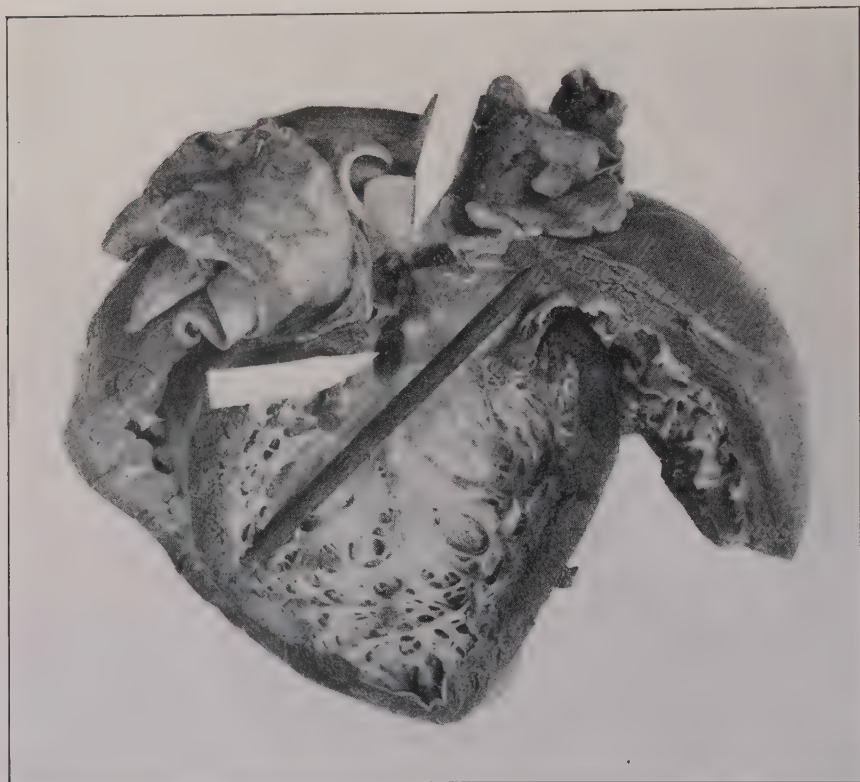


FIG. 49.—DEFECTIVE INTERVENTRICULAR SEPTUM AND BICUSPID AORTIC VALVE IN INFANT 7 WEEKS OLD.

(Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

being found in one-third of all autopsies performed upon adults, and is rarely diagnosed during life. It gives no symptoms because the valves usually close the slitlike orifice and because the pressure in the two auricles is so nearly equal. In dilatation of the heart it may become a larger orifice. The interauricular septum may be absent, giving one auricle, or may be partially developed with a large defect (Fig. 51). Only the advanced degree is serious. Open foramen ovale is important

as a means of passage of thrombi from the veins into the arterial circulation, which is hard to explain by any other mechanism.

PULMONIC STENOSIS.—Malformations of the orifices of the large vessels depend on anomalies of the septum intermedium or aortic septum. Pulmonic stenosis is rare without septal defect or other lesions, but moderate degrees occur (Fig. 52). The extreme degrees are incompatible with life without compensating lesions.

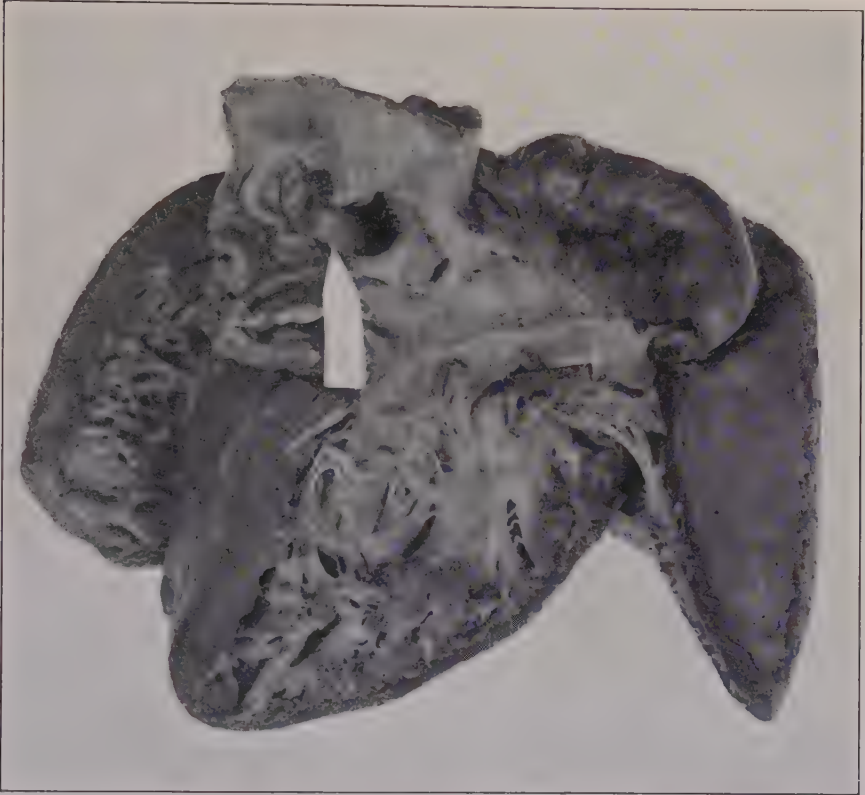


FIG. 50.—SAME HEART AS IN FIGURE 49, SHOWING PATENT FORAMEN OVALE.
(Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

PATENT DUCTUS ARTERIOSUS.—The duct of Botalli may remain open as a single lesion, but is more often combined with others (see Figs. 48 and 52). It has been involved in ulcerative endocarditis, and even by an aneurysm. It may become thrombosed and give off emboli. As a rule, its patency causes no symptoms.

Combined Lesions.—Combined lesions are much more common than any of the single lesions mentioned. As a matter of fact, it is

often the compensating effect of one lesion which makes life possible in the presence of another of marked degree. Stenosis of the pulmonary orifice may be extreme, even amounting to atresia, the aorta being connected with both ventricles and the pulmonary arteries receiving all their blood through the patent ductus arteriosus.

Stenosis of the conus pulmonalis varies in degree with hyper-



FIG. 51.—DEFECTIVE INTERAURICULAR SEPTUM.

(Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

trophy of the right ventricle when marked and often with septal defect. The ductus may be open or closed. There may also be present defect of auricular septum and transposition of the aorta and pulmonary artery or aorta given off from both ventricles due to a high septal defect.

The most common congenital malformation compatible with life is moderate pulmonic stenosis with septal defect. Hypertrophy of the right ventricle may give adequate circulation. Cyanosis results when

the circulation is insufficient. In advanced stenoses a vicarious blood supply to the pulmonary arteries may come from the dilated mediastinal, pericardial, esophageal and bronchial arteries. A patent ductus is the best collateral circulation. Few cases of marked stenosis live beyond puberty.

Stenosis of the aorta, a rare lesion, may be at the orifice. It is



FIG. 52.—CONGENITAL PULMONARY STENOSIS IN INFANT, 2 MONTHS OLD, WITH PATENT FORAMEN OVALE AND PATENT DUCTUS ARTERIOSUS, INDICATED BY ARROWS.

(Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

usually combined with other lesions (patent ductus). Isthmus stenosis, in the region between the left subclavian and the ductus, is not so rare. This may be a narrow tubular constriction with a patent ductus or a funnel-shaped narrowing and a closed ductus. In the latter case the circulation is carried on through the subclavian and vessels of the chest wall to the anastomoses with the branches of the descending

aorta. The left ventricle, or even the entire heart, becomes hypertrophied. Bicuspid aortic valve is often associated with coarctation of the aorta (Figs. 53 and 54).

Dextrocardia occurs rarely without complete situs inversus of the viscera, but the latter condition is seen fairly often. It is often overlooked in life, especially in young infants. It is necessary to keep this



FIG. 53.—BICUSPID AORTIC VALVE IN 3 MONTHS OLD INFANT.

This condition is often associated with coarctation of the aorta. (Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

possibility in mind in examining every child and carefully examine the right side for signs indicating a right-sided heart. We have seen one case of definite situs inversus with signs which suggested pulmonic stenosis. The murmur was loudest to the right of the sternum and the first diagnosis was aortic stenosis.

The association of congenital heart disease with mongolian idiocy is frequent enough to be noteworthy. No explanation has been offered

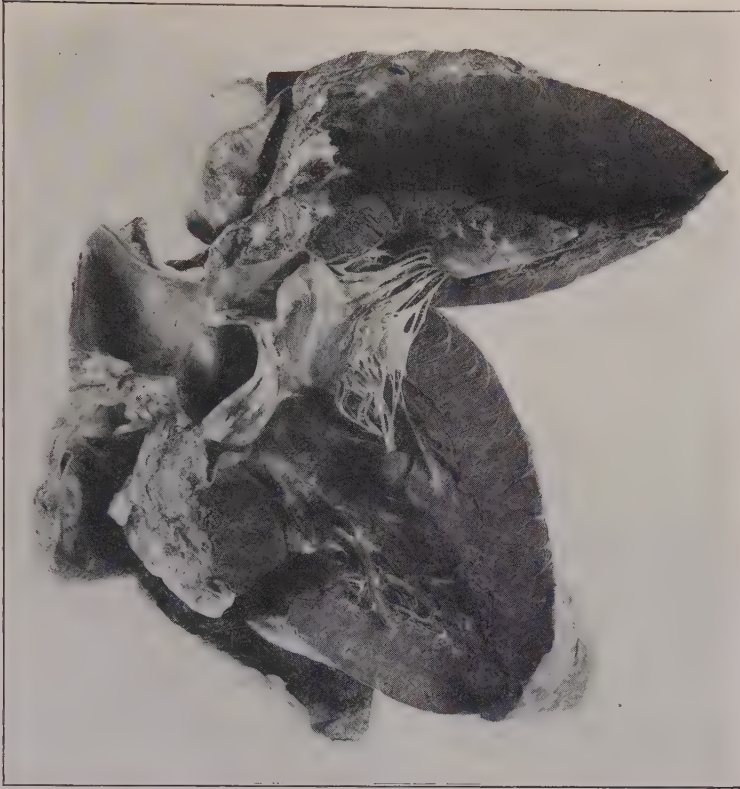


FIG. 54.—BICUSPID AORTIC VALVE, IN BOY, AGED 16 YEARS.
(Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

except that both are developmental faults. This association perhaps points away from the endocrine origin of mongolism.

Still gives the following incidence of congenital lesions:

Septal defect.....	20	(only lesion in 4)
Patent ductus.....	5	
Patent foramen ovale.....	1	(widely open)
Pulmonic stenosis.....	15	(only lesion in 6)
Pulmonic atresia.....	3	
Aortic stenosis or fusion of valves	12	
Tricuspid stenosis.....	2	

CLINICAL VARIETIES

Classification.—Cases of congenital heart disease may be divided into three groups :

1. Those with lesions so severe that the circulation is greatly impaired and life is impossible. These infants are deeply cyanosed from birth and live only a short time. They include the two- or three-chambered hearts, enormous septal defects, or marked stenosis of a pulmonary valve, atresia of the aorta, and so forth. Those who do live for a few days or weeks have great difficulty in breathing and can hardly nurse on account of the dyspnea and abnormal circulation. Such cases are often complicated by intracranial hemorrhage, and frequently have other congenital abnormalities.

2. Less severe cases who are cyanosed but who do live on for months or even years. They have more or less dyspnea, get very blue on slight exertion, crying or even on nursing.

In the milder cases the general development is only moderately affected. The child usually develops slowly and may behave like a difficult feeding case but gradually does gain, although he never catches up to the average. He may be blue all the time or only after exertion. The fingers and toes become clubbed if he lives long enough. The heart action may be tumultuous on slight effort.

In some children the cyanosis gradually disappears and they do very well. In others it persists and the child goes on as a semi-invalid, never able to do very much. It is surprising, however, to see how well some of these children get along, even with severe cyanosis and almost constant dyspnea.

3. Cases with definite congenital lesions without cyanosis. This is a large group. The children seem to suffer very little, if any, inconvenience from their lesion. In a few who have been observed from infancy there has been a slow gain in spite of good general health. Later these children have picked up and have done very well. The majority of the cases without cyanosis are apparently perfectly normal children. The lesion is definite enough to give physical signs, but does not affect the circulation appreciably and hence does not trouble the child in any way. These are usually septal defects, patent ductus arteriosus or even slight pulmonic stenosis. Some of the combined lesions give no symptoms, although the murmurs are loud and complex, and one listening to the heart can scarcely believe that it is able to function normally.

The sign which is of greatest help in making the prognosis is the degree of cyanosis. Without this sign one can almost surely promise a good outcome even with loud murmurs. If only slight cyanosis occurs after crying the prognosis is not wholly bad. Cardiac enlargement occurs in the worst cases and is an unfavorable sign.

The clinical importance of congenital malformation of the heart is twofold: Differentiation from rheumatic heart disease, and the fact that congenital defects are apt to become the site of acute endocarditis, either rheumatic or subacute bacterial in origin. The actual incidence of congenital heart disease severe enough to give signs and symptoms is probably not high. In this chapter will be described the physical signs which are commonly found with the most frequent congenital defects. It must be pointed out, however, that the signs are not necessarily diagnostic of the lesion present. Antemortem and postmortem diagnoses of the defect often do not coincide. In many cases there are no physical signs, and the lesions are only found after death. In the presence of signs, it is usually not difficult to be sure that there is a congenital malformation, but it is difficult to be sure what the defect is. There are almost always multiple defects, and this adds to the confusion in interpreting the physical signs. The illustrations of pathological specimens show clearly that more than one defect is usually present.

Single Lesions.—PULMONIC STENOSIS.—This is one of the more



FIG. 55.—PULMONIC STENOSIS.

Heart

Apex: Fifth space inside nipple 2 inches to left.

Action: Regular, slow, forcible.

Sounds: Pulmonic second very faint.

Murmurs: Systolic blow over most of left side. Maximum at pulmonic area and up to first space, where it is also rough in quality.

Thrill: Marked over maximum site of murmur.

frequent lesions found in congenital heart disease, ranking second only to septal defect and being one of the most common of the combined lesions. It is frequently associated with patent foramen ovale, septal defect and patent ductus arteriosus. Many of the children with this lesion grow up with little or no disability. In the severer grades there may be cyanosis, dyspnea, clubbing, and so forth.

The size of the heart is not usually much increased, though there is some hypertrophy of the right ventricle. This rarely gives appreciable enlargement clinically. The action of the heart is not affected in the average case.



FIG. 56.—PULMONIC STENOSIS.

The sounds are characterized by a diminution or absence of the second pulmonic sound, which is a striking sign in childhood because the second pulmonic is normally louder than the aortic sound. The feeble or absent second pulmonic sound is due to the lessened blood-pressure in the pulmonic circulation due to the stenosis. The murmur is systolic, usually rough or harsh and loud. It is loudest over the second left space and transmitted best up to the first (Fig. 55), but may be heard over a fairly wide area when loud (Fig. 56). There is often a marked thrill.

The diagnosis is made by the rough systolic murmur and thrill over the site described with weakening of the second pulmonic sound.

The prognosis depends on the degree of narrowing. Many children with the characteristic signs go on for years with few or no symptoms. The severer grades, with symptoms and poor heart action, have a bad prognosis.

DEFECT OF THE INTERVENTRICULAR SEPTUM OR SEPTAL DEFECT.—This is the most common of all single congenital heart lesions. The term “patent septum” is a poor one and should not be used, since “patent” implies a persistence of an opening present normally in fetal life. The signs of septal defect vary a good deal, depending on the size

of the defect and probably also on its site. The usual site is high up just below the fibrous part of the septum near the auriculoventricular ring.

The disturbance in the circulation depends largely on the amount of blood which takes an abnormal course. If the pressure in the two ventricles were absolutely equal the defect would make little difference, but since the left ventricle is stronger some blood is forced into the right ventricle in systole. It is difficult to see why this should do any great harm. It may be that in the large defects the venous blood from the right ventricle flows into and mixes with that in the left during diastole. This is perhaps the best explanation for the cyanosis seen in the severe cases.

The smaller defects give no symptoms whatever, although the murmur may be very loud.

The moderate defects may give some dyspnea on exertion and disturbed heart action, but the individual often goes through life with little or no discomfort. The large defects give the usual symptoms of dyspnea on the slightest exertion, or even at rest. The infant may be cyanosed from birth and may live a few hours or a few months. If he lives longer there is clubbing of the fingers and toes, and the child is frequently stunted and undernourished. Few of these children grow up but they may drag out a miserable existence up to puberty. Intercurrent acute disease is badly borne.

The heart is seldom enlarged except in the worst cases and then only moderately so. In the mild cases the action is perfectly forcible, regular and little affected by exercise. The sounds may be normal or the first sound may be largely obscured by the murmur. The murmur is systolic, usually extending through all of systole. It is maximum over the region just inside the apex, or along the left sternal border,



FIG. 57.—SEPTAL DEFECT.
This boy had no symptoms.

or even to the right of the sternum (Fig. 57). It is not often heard outside the apex, except when extremely loud. The total area varies with the intensity, but in general is over the precordium tending to extend to the right of the sternum. When loud the murmur is audible over all of the chest and often through to the back (Fig. 58), but is rarely heard to the left of the precordium. This is an important differential point between septal defect and mitral insufficiency. The quality of the murmur varies from a soft blow to a harsh whistling sound. Its intensity also varies. Some of these murmurs are very loud, others only moderately so. There may be a thrill over the precordium but this is not often widely felt.

This lesion must be differentiated from mitral insufficiency, pulmonic stenosis and functional systolic murmurs. It is not always easy to be certain of the diagnosis.

The differentiation from a mitral lesion cannot be made on the age unless the child is seen in early infancy, since mitral lesions do occur very early (eighteen months). The mitral murmur may have exactly the same distribution but is transmitted to the left of the apex in the majority of cases, and is usually louder at the apex than inside it. History of rheumatic polyarthritis or chorea always suggests an acquired lesion, but the absence of a definite history does not rule out rheumatic carditis.

PATENT FORAMEN OVALE.—A patent foramen ovale is almost normal, since it must close after the cessation of the fetal circulation. The persistence of this patency seems to give no great trouble because the pressure in the two auricles is so nearly alike that no great amount of blood flows through the foramen. There are no diagnostic symptoms nor signs of the persistence of this opening.

Small openings usually give no signs, and even large openings may not be suspected before death. The symptoms are those found in the presence of any congenital defect, namely, cyanosis, dyspnea, poor nutrition and cold extremities. Other congenital malformations are almost always present and these associated lesions are probably responsible for the murmurs present, for the interference with the circulation, and hence for the symptoms.

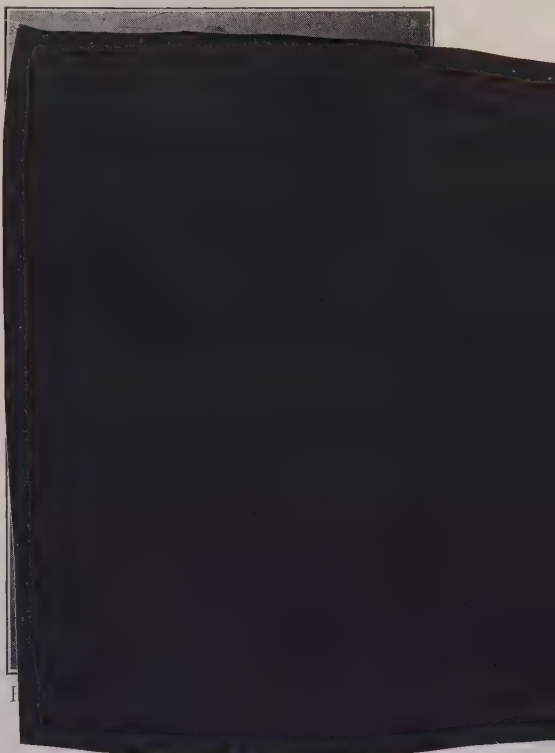
PATENT DUCTUS ARTERIOSUS.—The ductus arteriosus which normally short-circuits the blood from the lungs in fetal life may remain patent. In that case blood is forced through it in systole by the ventricular contraction and during diastole, on account of the higher blood-

pressure and stronger elastic wall of the aorta. This lesion causes less disturbance to the circulation than most of the other forms of congenital heart disease, since the duct is rarely wide enough to allow the passage of large volumes of blood. In the Huddleston Class no case of cyanosis has been noted with this lesion, except when combined with others. The patients having it are ordinarily quite unconscious of their hearts, the murmur being discovered by accident. We have seen one medical student with this lesion who played football all through college. Cyanosis and clubbing, which are so common in other defects, are unusual. There may be dyspnea on exertion.

The diagnosis can be made by the physical signs. The murmur is very loud and harsh, and often has the characteristic "coffee mill" sound. Although it usually is both systolic and diastolic in time (a continuous murmur), it may be chiefly systolic (Fig. 59). The pulmonic second sound is greatly accentuated, which helps to distinguish the condition from pulmonic stenosis. The murmur is transmitted upward to the left carotid, and may be heard in the left interscapular region.

Combined Congenital Lesions.

—The diagnosis of combined congenital lesions is always presumptive. Combined lesions are much more frequent than any single lesion. But the signs are often so difficult to dissociate that it may be impossible to be sure that there is a double lesion. The most common is a combination of pulmonic stenosis and septal defect. In this group the pulmonic stenosis is the striking lesion. The systolic murmur runs into



Heart

Size: Apex diffuse, maximum in fifth space, $2\frac{5}{8}$ inches to left, palpable to 3 inches to left. Right border $\frac{1}{2}$ inch to right of sternum.

Action: Regular, forcible, slow.

Sounds: First partly obscured by loud murmur, second sound equal.

Murmurs: Loud, harsh, systolic, heard over whole chest, maximum along left sternal border, as indicated.

that from the septal defect and the presence of the latter can only be inferred.

A combination of patent ductus and pulmonic stenosis is again difficult because the systolic murmur from each has its maximum site at about the same place.

We see some cases of congenital heart disease in which no diag-



FIG. 59.—PATENT DUCTUS ARTERIOSUS.

Heart

Apex: Maximum in fifth space, $2\frac{1}{2}$ inches to left, palpable to $2\frac{3}{4}$ inches.

Action: Excellent, regular, forcible, slow.

Sounds: At base obscured by murmurs, but second aortic louder than pulmonic.

Murmurs: Systolic blow as indicated by dots. Maximum over second left space and down left sternal border.

Diastolic as outlined by dashes. Maximum over second left space.

Two murmurs make a continuous loud roar with systolic accentuation.

nosis can be made. It is evident that the disease is congenital but the signs do not correspond to any of the recognizable lesions. One such boy has been deeply cyanosed from birth, has dyspnea constantly, increased by the least exertion, though he has been able to go to school. He was seen first at three years. His heart was not enlarged but was overacting. There was a very short, faint systolic murmur at the base. Along the left sternal border there was a peculiar midsystolic

clicking sound. This has persisted until the present, when he is eleven years old. The examination of his heart was recorded as follows:

Size: Enlarged; apex-beat diffuse, of fair force, maximum in fifth space $4\frac{3}{4}$ inches to left, palpable to $5\frac{1}{4}$ inches to left of midline.

Left border corresponds.

Right border percusses $1\frac{1}{2}$ inches to right.

Action: Regular, rather overacting.

Second pulmonic sound accentuated.

No murmurs heard.

There is a peculiar clicking sound heard in middle or late systole after the rather short first sound. This is audible over most of the precordium, loudest along the left sternal border.

The electrocardiogram showed merely some right preponderance and the x-ray showed right hypertrophy more than left.

Many specialists have seen this boy (a private patient) and no one can make a diagnosis. He may have a very wide defect in his septum without murmur, or some anomalous distribution of the blood-vessels. This child has survived several acute infections, pneumonia, tonsillitis, measles. He has been very sick with all of them. He is thin and has poor muscles, has a very difficult temperament but still lives on, goes to school, keeps up with his class in spite of his almost black color on very slight exertion.

We have another group of congenital hearts under observation not corresponding with any of the easily recognized lesions. All the signs of pulmonic stenosis are present except that the murmur is atypically situated, being loudest to the right of the sternum in the second space and transmitted up and to the right only a little way. There are none of the signs of aortic stenosis, little or no hypertrophy. The pulse is good. There is not much thrill.

Whether these are cases of pulmonic stenosis with an unusual site for the murmur, or slight congenital narrowing of the aorta, or some other condition, we are in doubt. After seeing several of them one feels that it is some unusual lesion, with characteristic signs. We have had no opportunity to determine the lesion at autopsy.

Other Rare Defects.—**COARCTATION OF THE AORTA.**—This is a constriction of the part of the aorta which lies between the left subclavian artery and the insertion of the ductus arteriosus. It is usually associated with other defects such as septal defect, pulmonic stenosis and bicuspid aortic valve. There may or may not be physical signs.

True DEXTROCARDIA is the result of transposition of all the viscera, and is not clinically significant, as no symptoms are present. It is found accidentally during examination. Transposition of the heart alone is very rare. When it occurs, the great vessels may be disarranged so that symptoms of congenital heart disease are produced.

Congenital disease of the tricuspid and mitral valves is extremely rare.

CHAPTER X

X-RAY EXAMINATION OF THE HEART

The value of x-ray examination of the heart lies in the information gained as to the size and, to a lesser extent, the shape of the heart. The contour of the cardiac shadow varies considerably in health as well as in disease in different individuals. The shape of the heart in adults can be correlated with the habitus of the individual, *i.e.*, the heavily built individual has a short broad heart; the thin, asthenic person has a long narrow heart; "drop" heart is found in the hyposthenic individual, while the commonest type of heart is associated with the sthenic habitus. The hearts of children between two and thirteen years of age fall into similar groups according to shape, but apparently these do not correspond to the habitus as they do in adults. The commonest shape is the sthenic type of heart, which Lincoln and Spillman found in 166 of 246 children of this age group studied. The hyposthenic is like the sthenic but narrower, and is relatively uncommon. They found the asthenic type in only one case, and the hypersthenic occurred only in children under five years of age (Figs. 60 to 63). It is possible that the type of a heart has some influence on the shape which it takes when diseased.

There are two methods by which the actual size of the heart can be measured: one by taking a two-meter film (teleroentgenogram), the other by making an orthodiagram.

When the patient stands or lies close to the immovable tube, the rays hit the heart at a tangent, so that there is a measurable magnification of the shadow. If he stands at a distance of two meters, the rays are approximately parallel, and the shadow thrown gives a true outline of both the size and the shape of the heart. The pulmonary fields are also clearly shown.

To demonstrate that the shadow of the heart cast when the tube is a distance of two meters from the patient is the same size as the heart itself, LeWald made a teleroentgenogram of a chest. The heart was then removed, and a photograph made the exact size of the heart. This photograph was then superimposed on the x-ray film, and the shadow of the heart in the film coincided exactly with the picture of

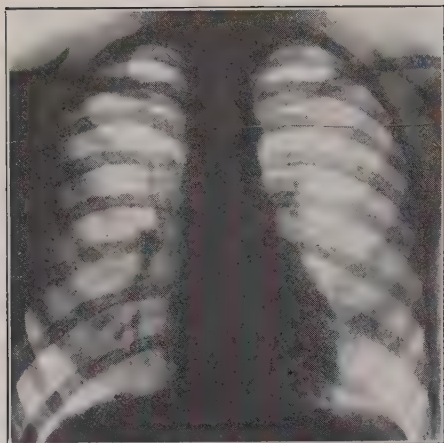


FIG. 60.—HYPOSTHENIC TYPE OF NORMAL HEART.

(Lincoln and Spillman, *Am. J. Dis. Child.*, Chicago, 1928.)

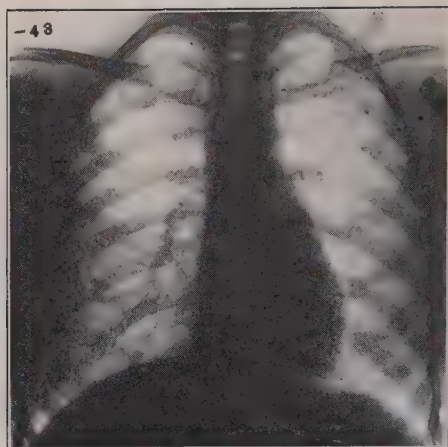


FIG. 61.—ASTHENIC TYPE OF NORMAL HEART.

(Lincoln and Spillman, *Am. J. Dis. Child.*, Chicago, 1928.)

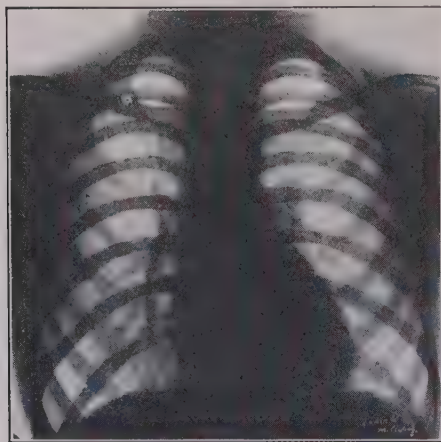


FIG. 62.—STHENIC TYPE OF NORMAL HEART.

(Lincoln and Spillman, *Am. J. Dis. Child.*, Chicago, 1928.)

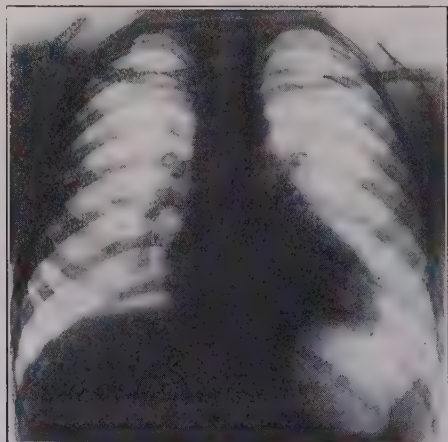


FIG. 63.—HYPERSTHENIC TYPE OF NORMAL HEART.

(Lincoln and Spillman, *Am. J. Dis. Child.*, Chicago, 1928.)

the heart. This is shown in Figure 64, and is proof that measurements of the heart taken from a two-meter plate are the true measurements of the size of the heart. This figure and Figure 65 show also the relationship of the heart and valves to the anterior chest wall.



FIG. 64.—RELATIONSHIP OF THE HEART AND ITS VALVES TO THE ANTERIOR THORACIC WALL.

Compare Figure 65; exposure made at a distance of 6 feet (1.8 meters); measurements verified later by dissection. (LeWald, *Arch. Surg.*, Chicago, 1923.)

The orthodiagram is made by a special attachment to a fluoroscope in which the carrier of the tube is movable. The patient stands close to the machine with the fluoroscopic screen just touching the chest. A point is marked on the screen, and the screen is moved so that this point follows the outline of the heart. The tube moves at the same time. A pencil is attached at the side of the screen, so that as the screen

and tube move, the outline of the heart and chest is marked on a piece of paper hanging in front of the pencil. When the examination is finished, the paper is removed, and the measurements of the heart and pulmonary fields are made from this tracing. The advantage of using

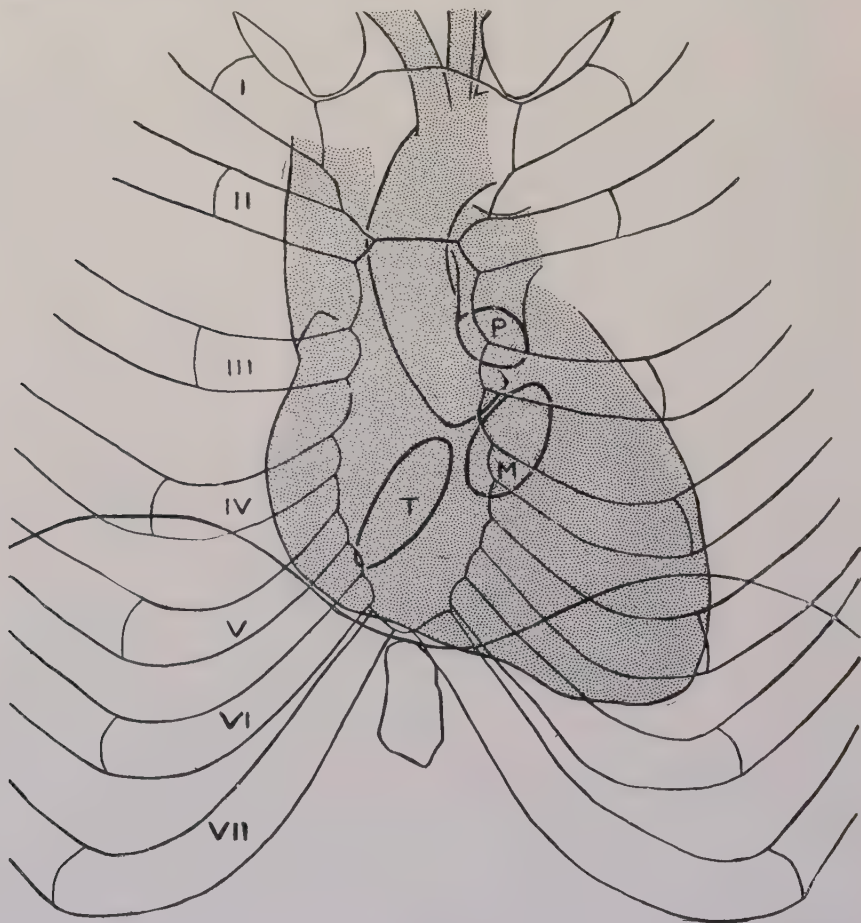


FIG. 65.—OUTLINE DRAWING OF TELEROENTGENOGRAM (FIG. 64) AND STEREOSCOPIC ROENTGENOGRAMS.

The position of the valves *T* (tricuspid), *M* (mitral) and *P* (pulmonary) may be noted. The aortic valve is indicated by the beginning of the aorta. (LeWald, *Arch. Surg.*, Chicago, 1923.)

the fluoroscope is that the examiner sees at first hand the movements of the heart, whether or not the apex is fixed during change in position or respiration and any unusual pulsations which may be present. The technic is very simple and can be acquired by anyone, but there is a certain facility which comes with practice. The tracings obtained are

surprisingly accurate. The procedure is suitable for clinic use, and even small children can be examined in this way. The orthodiagram is also well adapted to office practice.

To take the measurements, a straight line is drawn down the center of the figure. At the level where the left side of the heart is widest, another line, at right angles to the first, is drawn from the vertical line to the border. Similarly, a line is drawn on the right side. The sum of these two lines is the greatest transverse diameter

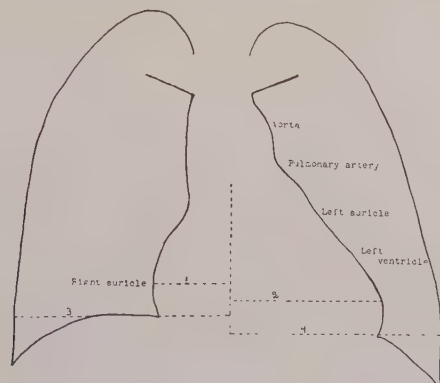


FIG. 66.—MITRAL INSUFFICIENCY—CLASS I.

Orthodiagram of L. M.

1 plus 2 equals maximum transverse diameter of heart—10.2 cm.

3 plus 4 equals maximum width of pulmonic fields—18.2 cm.

of the heart. Lines are drawn at the level of the diaphragm on each side, from the midline to the internal edge of the ribs. The sum of these lines equals the width of the pulmonary field. The transverse diameter of the normal heart is not more than half the width of the pulmonary field. Normally about one-quarter to one-third of the heart lies to the right of the midline. The longitudinal diameter is of little value. Figure 66 demonstrates the method of making these measurements.

Accurate determination of the size of the heart by x-ray is most valuable in suspected mitral insufficiency where clinical examination does not clearly show enlargement. In aortic disease the enlargement is usually so great that no evidence is needed to make the diagnosis other than that found on physical examination.

The shape of the heart as shown by x-ray is often interesting but may be misleading. It not infrequently happens that the x-ray diagnosis of mitral stenosis is made on the shape of the shadow when

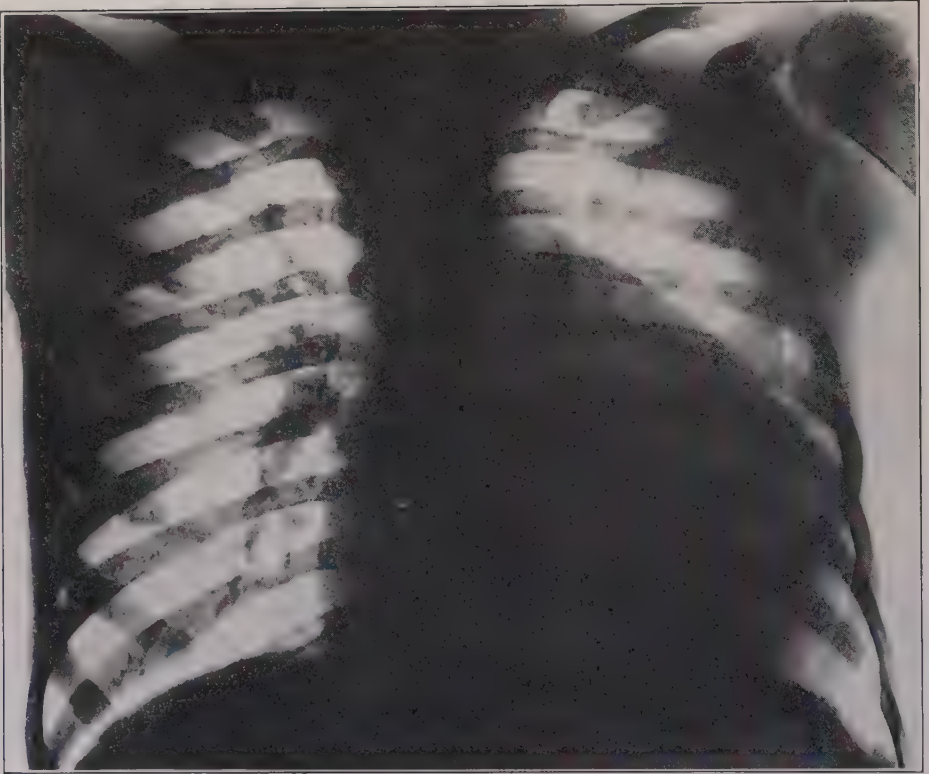


FIG. 67.—DOUBLE MITRAL LESIONS. ACUTE CARDITIS.

Possibly small amount pericardial effusion. Many rheumatic nodules. Lung signs on left posteriorly caused by compression.

X-Ray Report (J. M., male, aged 8 years)

No pathology of lungs.

Heart: Increased oblique cardiac axis. Triangular shape. Enlarged in all diameters. Accentuated right and left auricular curves and pulmonary artery.

Diagnosis: Enlarged heart. Double mitral valvular disease.

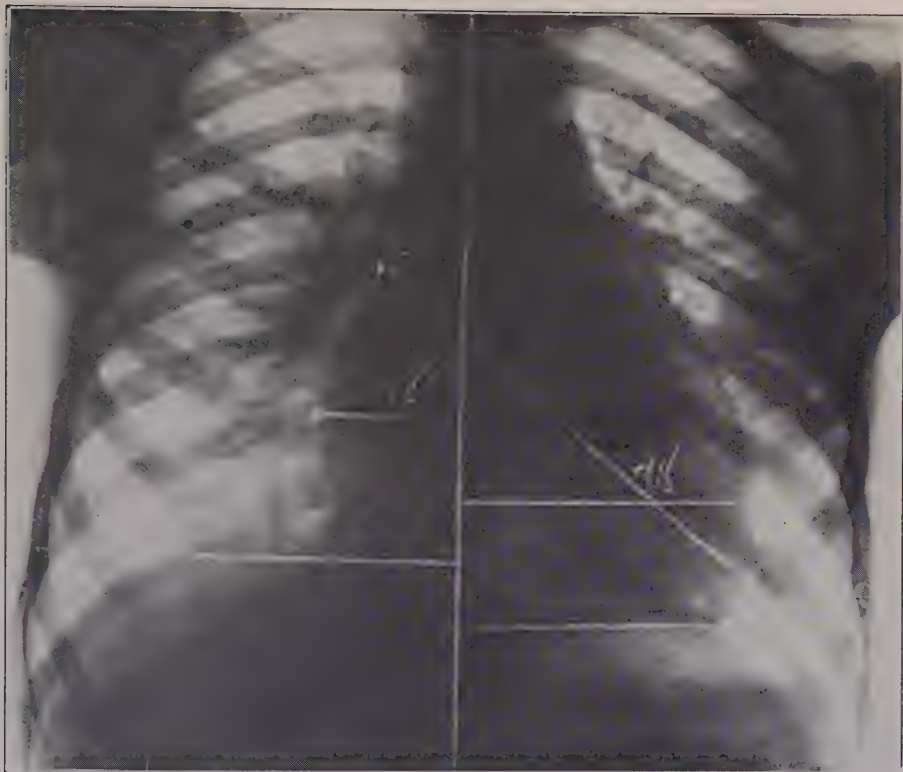


FIG. 68.—ACUTE CARDITIS. DOUBLE MITRAL LESIONS. RHEUMATIC PNEUMONIA.

Later had joint pains with heat and swelling and congestive failure.

X-Ray Report (G. M., male, aged 7 years)

Heart: Triangular. Enlarged in all diameters. Accentuation of right and left auricular curves.

Diagnosis: Enlarged heart. Double mitral valvular disease.

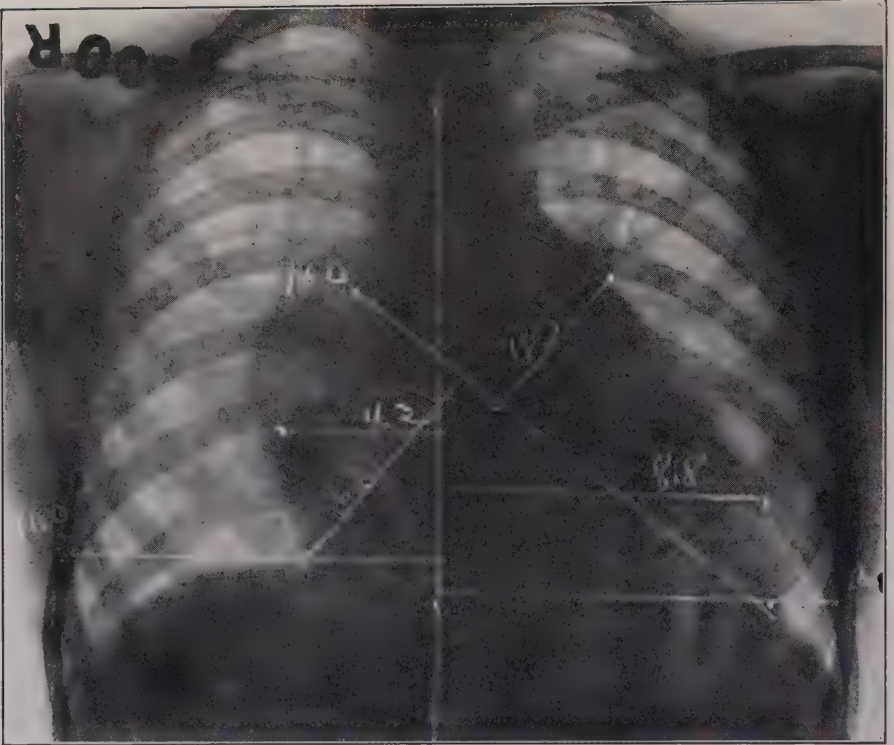


FIG. 69.—DOUBLE MITRAL LESIONS. ACUTE CARDITIS—CLASS 2B.

X-Ray Report (F. R., male, aged 8 years)

Heart: More vertical than normal. Triangular. Enlarged in all diameters. Accentuation right and left auricular curves, and pulmonary artery.

Diagnosis: Enlarged heart. Double mitral valvular disease.



FIG. 70.—DOUBLE MITRAL LESIONS, ACUTE CARDITIS. MANY RHEUMATIC NODULES.

X-Ray Report (J. L., male, aged 10 years)

Heart: Normal oblique cardiac axis. Triangular. Enlarged in all diameters. Accentuation right and left auricular curves. Obliteration cardiovascular angle.

Diagnosis: Enlarged heart characteristic of double mitral valvular disease.

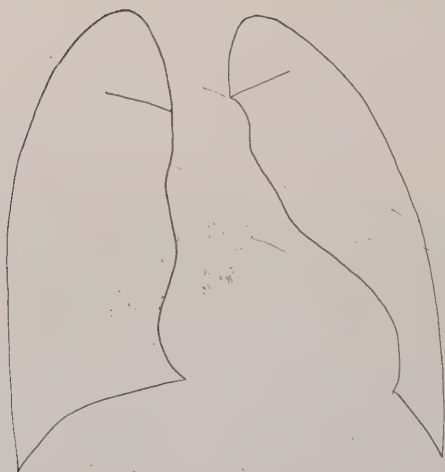


FIG. 71.—MITRAL INSUFFICIENCY AND STENOSIS—CLASS I.

Orthodiagram of M. N., February 14, 1928.

Apex-beat 6.5 cm. from midsternal line in fifth left interspace. Pulmonic fields 16.2 cm. Transverse diameter of heart 9.8 cm.

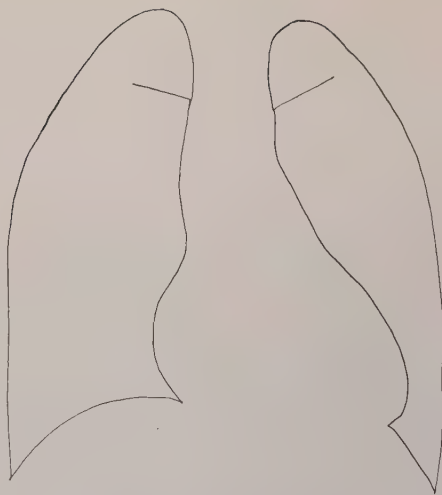


FIG. 72.—MITRAL INSUFFICIENCY AND STENOSIS—CLASS 2A.

Orthodiagram of M. N., March 7, 1928. Apex-beat 7.5 cm. from midsternal line in fifth left interspace. Pulmonic fields 16.3 cm. Transverse diameter of heart 9.1 cm.



FIG. 73.—MITRAL INSUFFICIENCY AND STENOSIS—CLASS 2B.

Orthodiagram of M. N., June 5, 1928.

Apex-beat 7 cm. from midsternal line in fifth left interspace. Pulmonic fields 16.7 cm. Transverse diameter of heart 9.7 cm.

Digitalis 0.05 gram, twice daily, started May 15th.

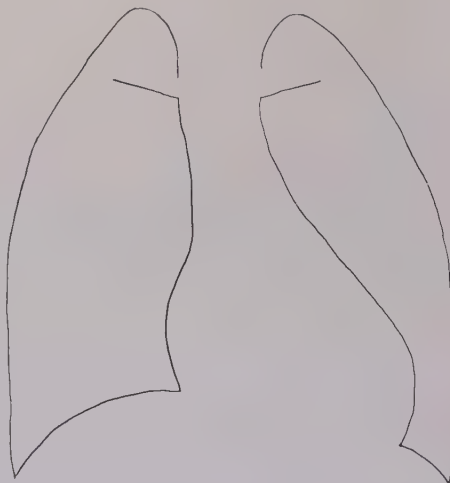


FIG. 74.—MITRAL INSUFFICIENCY AND STENOSIS—CLASS 2A.

Orthodiagram of M. N., September 25, 1928.

The shape of the orthodiagram suggests that the predominating lesion is now mitral stenosis. Pulmonic fields 16.5 cm. Transverse diameter of heart 9.0 cm.

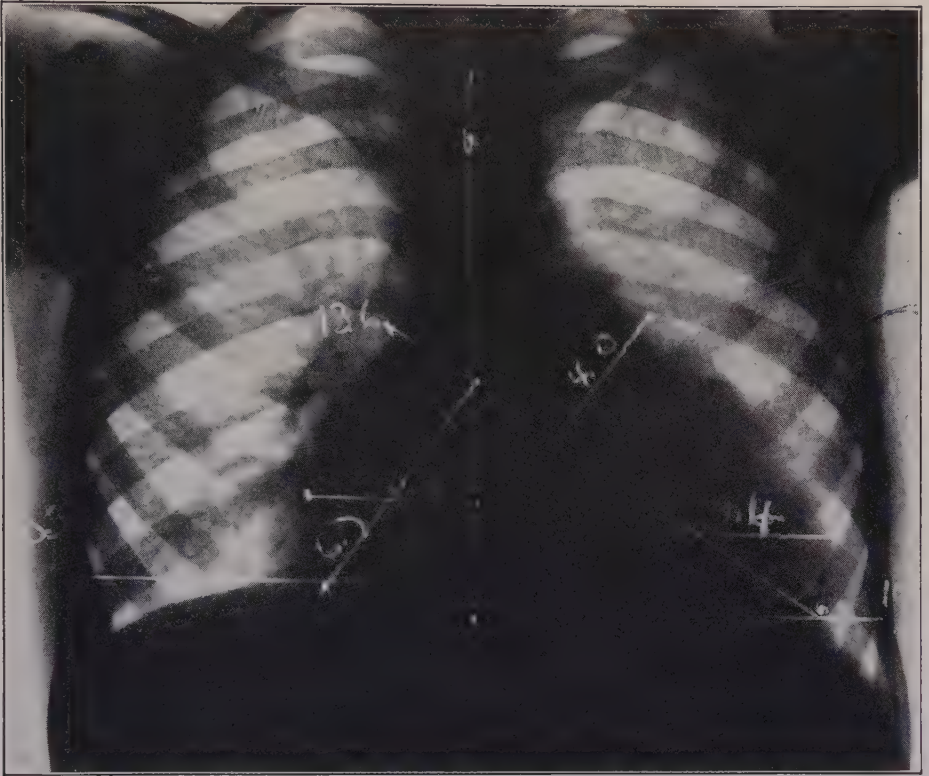


FIG. 75.—ACUTE CARDITIS. DOUBLE MITRAL LESIONS AND AORTIC INSUFFICIENCY.

X-Ray Report (I. M.)

Heart: More vertical than normal. Triangular. Enlarged in all diameters. Accentuation of all cardiac curves.

Aorta: No pathology.

Diagnosis: Enlarged heart.

there is no evidence of heart disease by physical examination. This diagnosis cannot be made in the absence of the characteristic murmur.

Figure 66 is an orthodiagram of the heart of a child with simple mitral insufficiency without stenosis. The measurements are given in this illustration and the method of making them is clearly shown. The heart is definitely enlarged; the transverse diameter is greater than half the width of the pulmonary fields. The great vessels and parts of the heart represented by the different curves of the outline are labeled. In advanced disease there may be rotation of the heart or enlargement of different parts so that the curves change.



FIG. 76.—MITRAL INSUFFICIENCY AND STENOSIS; AORTIC INSUFFICIENCY.

Orthodiagram of T. B.

Pulmonic fields 19.2 cm. Transverse diameter of heart 11.6 cm.

In mitral insufficiency there is some enlargement of the cardiac shadow, due to increase in size of the left and right ventricles. The contour of the left side does not change particularly, but the right side is exaggerated. In some cases pulsations are seen where the right contour touches the diaphragm. These are caused by the right ventricle.

In mitral stenosis the left side of the heart is enlarged, both auricle and ventricle (Figs. 67 to 70). The apex often seems to be pointed and may be pushed inward and downward. In slight stenosis the apex is rounded as in a normal heart. In advanced stenosis the left auricle is greatly enlarged. This is shown in the x-ray of J. L. (Fig. 70), a boy with double mitral murmurs, active infection of the myocardium, and many rheumatic nodules.

The four orthodiagrams of M. N. (Figs. 71 to 74), a seven-year-

old girl who was closely followed in the clinic, are interesting in showing the changes in the contour of the heart. Over a period of eight months, the stenosis became the predominating lesion, although the mitral systolic murmur persisted.

Aortic insufficiency produces hypertrophy of the left ventricle, and in children is usually seen in association with mitral disease. The x-ray of I. M. (Fig. 75) shows great enlargement of both sides of the

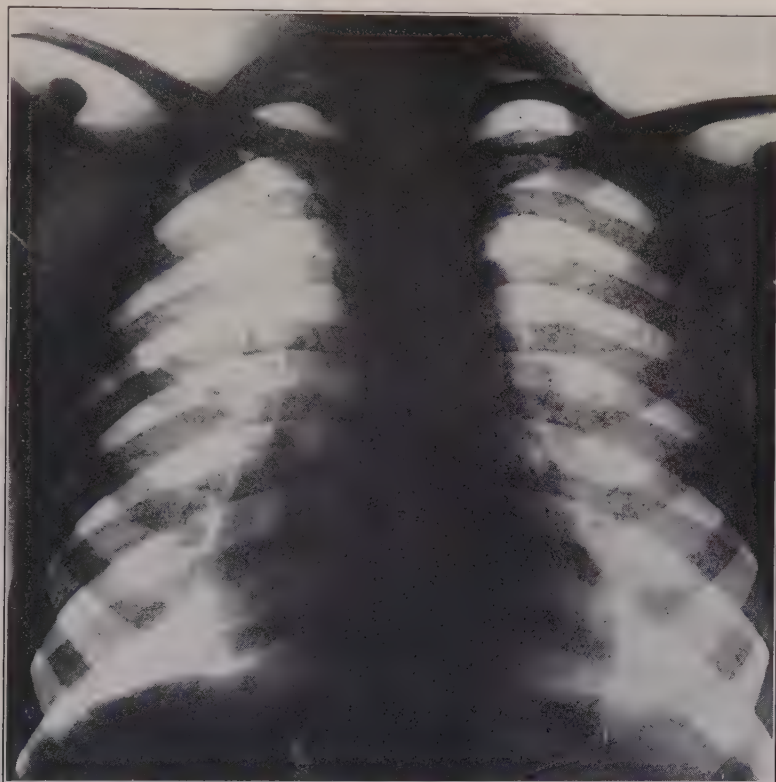


FIG. 77.—ACUTE MYOCARDITIS.

V. C., male, aged 8 years. First and second stage heart-block. Note right-side enlargement.

heart. He had active infection of the heart as evidenced by high irregular fever, increased leukocyte count, rapid heart rate and eventually a large crop of subcutaneous rheumatic nodules. It is interesting to compare this photograph with the orthodiagram of T. B. (Fig. 76), who had the same condition, *i.e.*, mitral insufficiency and stenosis and aortic insufficiency. I. M. has a short, wide thoracic cavity, with a broad, squat heart, while T. B. has a long narrow chest, with a

heart that was probably of the sthenic or hyposthenic type before it became diseased. Another factor was the presence of active infection in I. M., while T. B. was in Class 2a at the time the tracing was taken.

In myocarditis some dilatation is present (Figs. 77 and 78). This causes enlargement of the heart in all directions. In the presence of

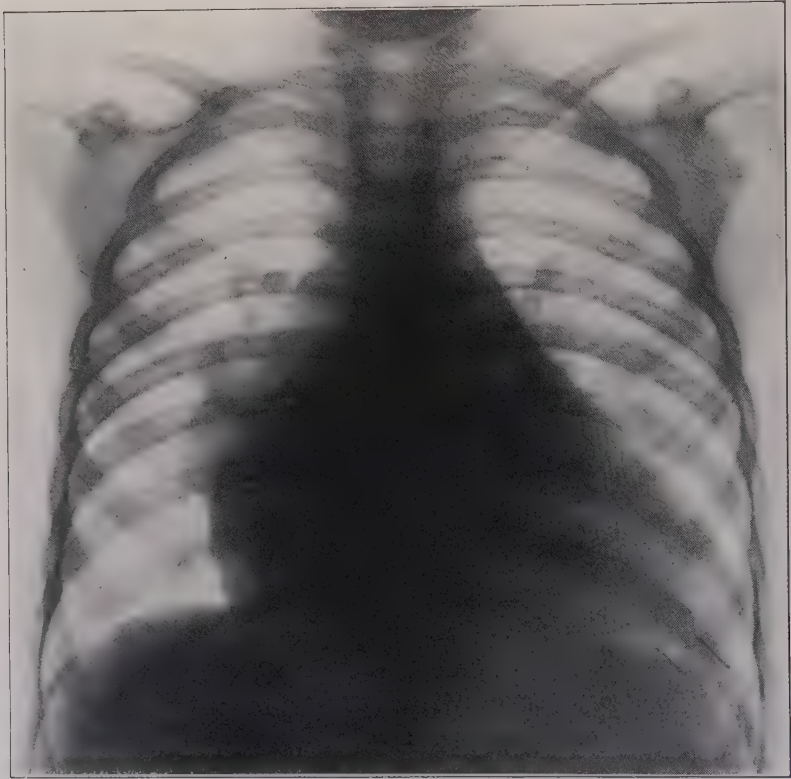


FIG. 78.—ACUTE CARDITIS.

S. C., female, aged 7 years. Double mitral murmurs. Many rheumatic nodules. Several attacks of acute fibrinous pericarditis and of congestive failure. Ill continuously for one year. Photograph of heart at autopsy (1 month after x-ray was taken) shown in Figure 15.

tricuspid insufficiency there is great enlargement of the right ventricle and auricle. Marked dilatation causes the heart to assume a triangular shape, with the base resting on the diaphragm. Sometimes the shape and enlargement in dilatation suggest the presence of pericardial effusion.

Pericardial effusion gives a globular increase to the shadow of the heart. The transverse diameter is greatly increased. The shape of the

cardiac shadow is characteristic. The shadow of the aorta is not changed, but below this there is marked enlargement to right and left. In large effusions the contour takes the "water-bottle" shape. Figure 79 shows a moderate pericardial effusion, and Figure 80 a large effusion. When the amount of fluid is small, the x-ray cannot give a

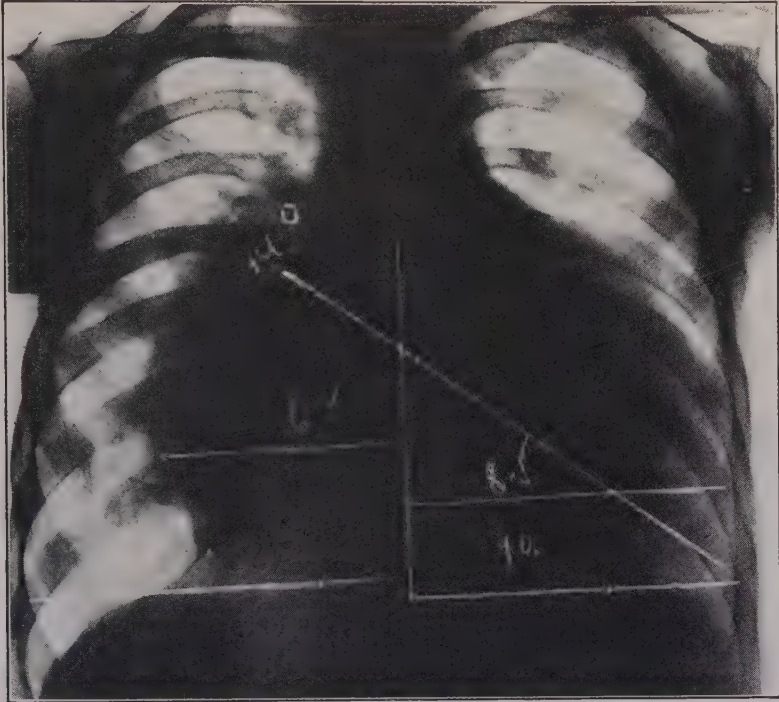


FIG. 79.—DOUBLE MITRAL LESION, ACUTE CARDITIS AND PERICARDIAL EFFUSION.

Heart apparently acutely infected all this time. Rheumatic fever continuously for one year.

X-Ray Report (A. H., female, aged 8 years)

Heart: Rounded. Enlarged in all diameters. Obliteration of cardiovascular angle.

Diagnosis: Enlarged heart. Pericarditis with effusion.

positive diagnosis, particularly if the heart itself is much enlarged and dilated. The pulsations of the heart in the presence of moderate or large effusions are usually diminished.

As we have pointed out in the chapter on physical signs, the presence of a moderate or large pericardial effusion may produce pulmonary signs on the left side. At times it is difficult to decide whether these signs are the result of pressure or of fluid in the pleural cavity. Such a case was J. H., a girl of seven years who had an enlarged, over-

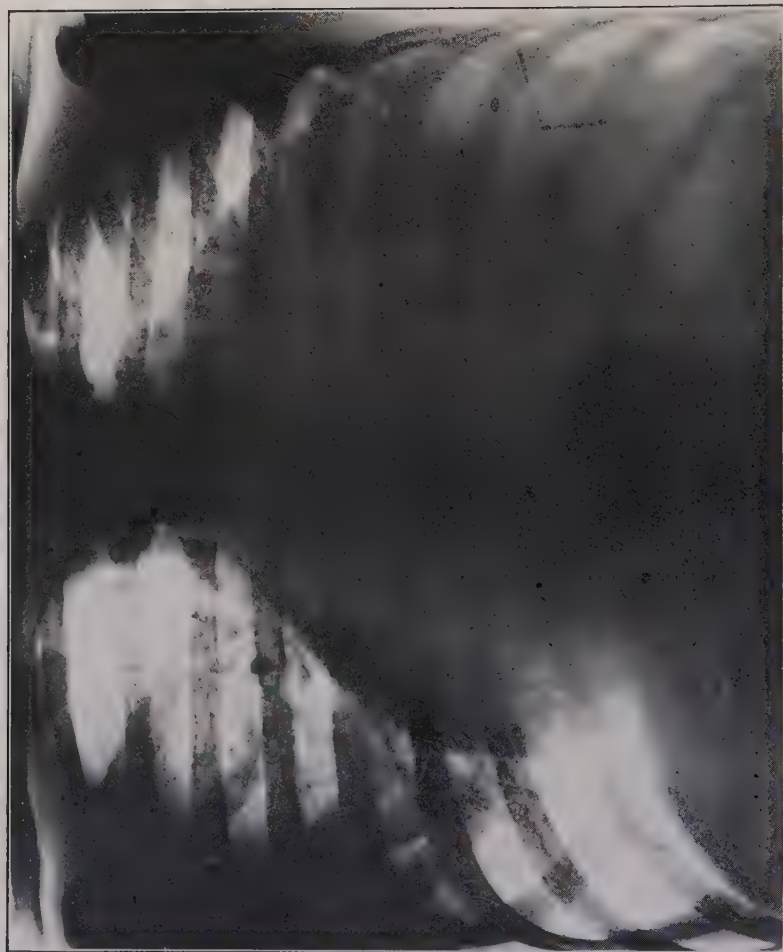


FIG. 80.—DOUBLE MITRAL AND TRICUSPID INSUFFICIENCY. PERICARDIAL EFFUSION. CONGESTIVE FAILURE. ASCITES. MARKED PULMONARY SIGNS CAUSED BY COMPRESSION OF LUNG BY ENLARGED HEART AND PERICARDIAL EFFUSION.

X-Ray Report (R. H., male, aged 11 years).
Heart: Round. Enlarged in all diameters.

Aorta: No pathology.
Diagnosis: Pericarditis with effusion.

acting heart, with flatness and greatly diminished breath sounds over the left lower lobe of the lung. Aspiration of fluid posteriorly proved nothing as to the source of the fluid, since this is the best point to aspirate pericardial fluid. The x-ray helped to make the diagnosis clear. Figure 81 shows a fairly dense shadow over the left lower lobe



FIG. 81.—MITRAL STENOSIS AND INSUFFICIENCY. RHEUMATIC PLEURISY WITH EFFUSION BEFORE TAPPING.

X-Ray Report (J. H., female, aged 7 years)

Left Lung: Pleural effusion which obliterates border of heart, dome of diaphragm and costophrenic sinus and extends upward in the axillary line to the third rib posteriorly.

Heart: Shadow widened in both diameters.

Diagnosis: Pleural effusion. Pericardial effusion.

with the upper level of the fluid indicated by the oblique line at about the fourth rib anteriorly and extending higher posteriorly. After removal of a large amount of fluid this shadow almost entirely disappeared (Fig. 82). It was clear that the fluid was in the pleura, not in the pericardium. This x-ray should be compared with that of R. H. (Fig. 80), who had extensive pulmonary signs at both bases, and who had a large pericardial effusion which was aspirated several times.

We feel that it is not wholly safe to make a diagnosis of particular valve lesions on the shape of the x-ray shadow alone. X-ray examination is of very great value in determining enlargement of the heart, especially when the enlargement is not great enough to be perfectly

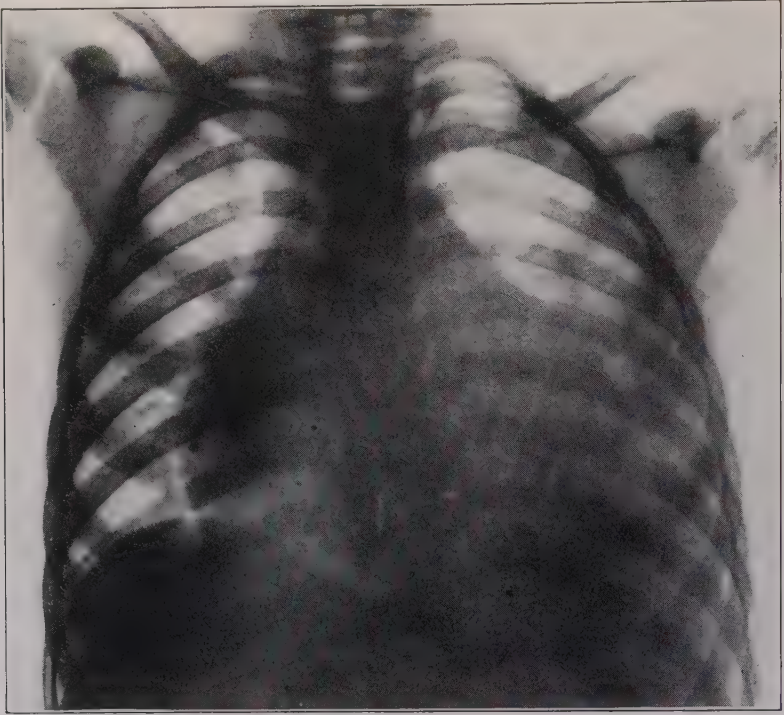


FIG. 82.—SAME CASE AS IN FIGURE 81. AFTER REMOVING PLEURAL FLUID.

clear clinically. As corroborative evidence in the different valve lesions the x-ray is a valuable aid. In differentiating between pleural and pericardial effusions, it is also of great importance when the clinical picture is confusing.

CHAPTER XI

ELECTROCARDIOGRAPHIC EXAMINATION

BY GERTRUDE NICOLSON, M.D.

History.—In 1856, Kölliker and Müller discovered that the contraction of a frog's heart generated an electrical current. On this basis, in 1887, Waller obtained records of the human heart, but since Eindhoven, in 1903, constructed the first string galvanometer, a vast amount of interesting facts has accrued concerning the mechanism of the heartbeat, making electrocardiography an integral part of modern cardiology. A record of precision is made through which the condition and progress of heart muscle and the toleration of medication may be accurately followed.

Value.—The electrocardiogram shows its value as a diagnostic aid in such problems as the differentiation of arrhythmias, the recognition of premature contractions with their site of origin, and in the diagnosis of doubtful "Steel" or "Flint" murmurs by showing the possible pulmonic, aortic or mitral origin of the condition. The electrocardiogram gives the only positive diagnosis of partial block, bundle-branch block, auricular flutter, paroxysmal tachycardia, nodal rhythm and acute coronary occlusion in the living subject.

Methods.—The construction and mechanism of the various electrocardiographs will not be described here. Those interested are referred to Sir Thomas Lewis's article on the string galvanometer in his *Mechanism and Graphic Registration of the Heart Beat*.

The procedure involved in making the record is simple and without disagreeable sensation on the part of the patient. An infant frequently falls asleep during the performance. Care must be taken to quiet a frightened child, as crying and movement may make the tracing useless. The child sits in a chair or lies on a stretcher, right and left upper and left lower extremities connected with the machine by electrodes. The small currents generated by the heart are led from the patient by means of wires to a delicate, silvered quartz fiber which moves at right angles to the current and to the magnetic field in which it lies. The shadow of this fiber is magnified and focused to a fine point so

that its deflections made by the cardiac currents may be standardized and recorded on a moving photographic film which becomes a permanent record.

Three leads are taken for a standard electrocardiogram. The first is recorded through the attachment of electrodes to the right and left upper extremities; the second to the right arm and left leg; the third lead connection is to the left arm and left leg. The interpretation of an electrocardiogram requires all three standard leads.

Interpretation of the Normal Electrocardiogram.—The study of the electrocardiogram of a diseased heart is based on a knowledge of the normal record so that deviations from its standards may be noted. The normal electrocardiogram (Fig. 83) shows a cardiac cycle consisting of a small upright deflection followed in a short space of time by a complex wave consisting of a part above the base line frequently immediately preceded and followed by smaller down strokes. Following this complex deflection at a greater distance than that of the preceding initial spike is a broad, rounded wave. The first of these deflections has been designated the P-wave. It alone corresponds to auricular action and is normally upright in all three leads. Its commencement forms the base line of the cycle and it normally precedes the succeeding deflection by an interval of not more than 0.2 second in time. This represents the time in which the stimulus passes from the pacemaker to the auriculoventricular node and is known as the P-R interval. The complex wave following is ventricular in origin and comprises the Q-R and S-waves. Q and S are the downward portions and either or both may be absent. The R-wave is usually the main ventricular spike and is always upright. The Q-R-S complex is a composite event lasting not more than 0.1 second and is composed of the superimposed right and left ventricular activities. In children, the Q-R-S interval may be notched or split, due probably to incoördination of the conduction and not to myocardial changes, as suggested in the adult.

The T-wave, which Lewis calls "stage of retreat" in ventricular contraction, is the final deflection in the cycle. This deflection should always be upright in the first and second leads. It is most variable in the third lead where its direction may change in the same person on such slight provocation as deep inspiration. It has been noted that the amplitude of this deflection in the second lead may be taken as a gauge of myocardial efficiency. An inversion of T in lead 2 has been shown to be significant of poor prognosis except in those instances

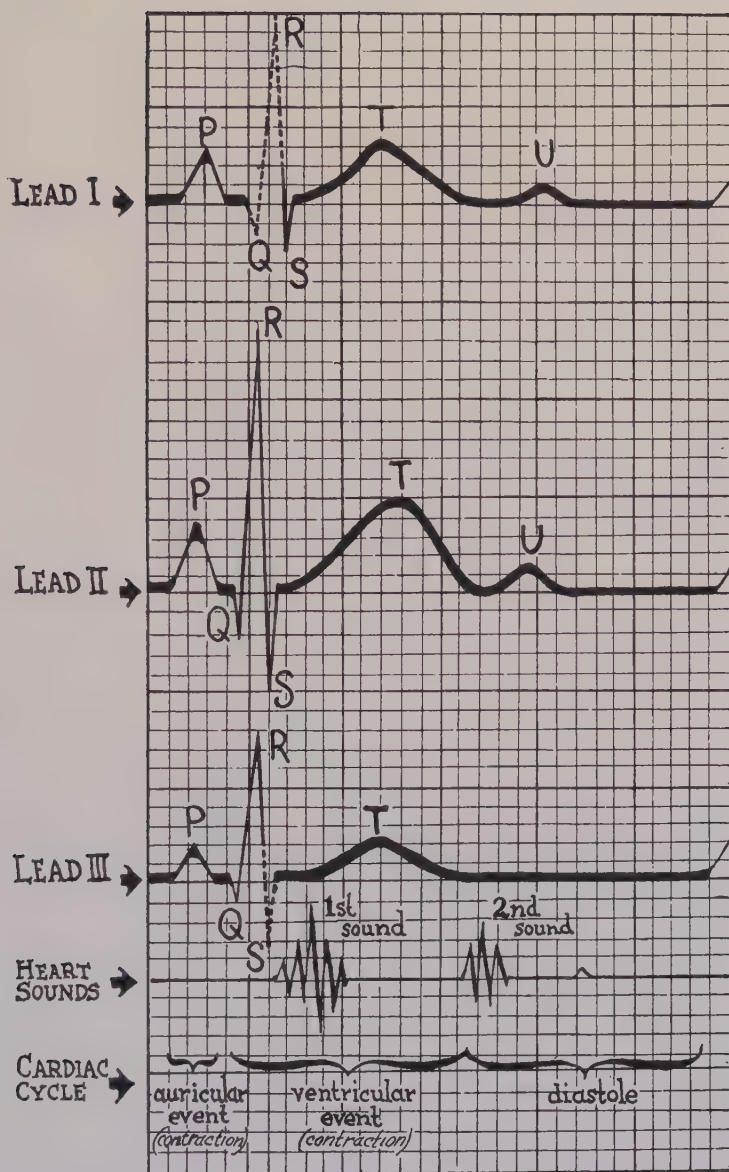


FIG. 83.—DIAGRAM OF THE THREE STANDARD LEADS OF THE ELECTROCARDIOGRAM WITH SOUND WAVE RECORD AND POSITION OF CARDIAC EVENTS.

Scale: vertical squares = 1 mvt.; horizontal squares = 0.04 sec.

where the condition is due to the effect of digitalis. In normal children the T-deflections are frequently of great amplitude and the "take-off" is occasionally as high as 1 millimeter from the base line.

Differentiation in the Child.—Normal standards have been laid down for the child's electrocardiogram through the studies of Nicolai and Funaro, Hecht, Krumbhaar and Jenks, Seham, Lincoln and Nicolson, and others.

The chief differentiation from the adult's electrocardiogram is noted in a greater amplitude of all the deflections and in the more rapid transmission of the excitation wave.

In the electrocardiogram of a cardiac child startling differences from the normal are not to be constantly expected, although fine changes, such as a decrease in amplitude of deflections and slight prolongation in transmission time, are not rare and are commonly overlooked. Thus, a typical clinical mitral stenosis or aortic lesion will frequently show no right or left ventricular predominance in the electrocardiogram and often disappoints the physician who wants positive evidence. On these grounds many clinicians fail to appreciate the extreme value of a positive finding by electrocardiography, not realizing that a correct diagnosis and prognosis are sometimes impossible without this instrument of precision, and also that an early electrocardiogram often aids in the interpretation of a later one. For instance, a case of acute chorea followed in a few months' time by a tightened mitral valve frequently shows a negative picture in the acute stage of the disease with an increasing tendency to right axis deviation as the lesion progresses.

Ventricular Predominance.—The *predominating* ventricle is found by examination of the first and third leads. A high R in lead 1 and deep S in lead 3 suggest left ventricular predominance (Fig. 84). A deep S in lead 1 and a high R in lead 3 indicate a preponderance of the right ventricle (Figs. 85 and 89). It may be mentioned here that Q₁, most of R₁, some of R₃ and S₃ are left ventricular events, while part of R₁, all of S₁ and Q₃ and most of R₃ show activity of the right ventricle (Fig. 83). Any marked increase of these divisions shows fairly definite predominance of the given ventricle. Thus, a right ventricular preponderance is marked in pulmonic stenosis (Fig. 93) and frequently in typical mitral stenosis (Fig. 85). A slight ventricular preponderance suggested by a single record does not settle a diagnostic question, and the condition should be noted in a series of electrocardiograms recorded over several months or years.

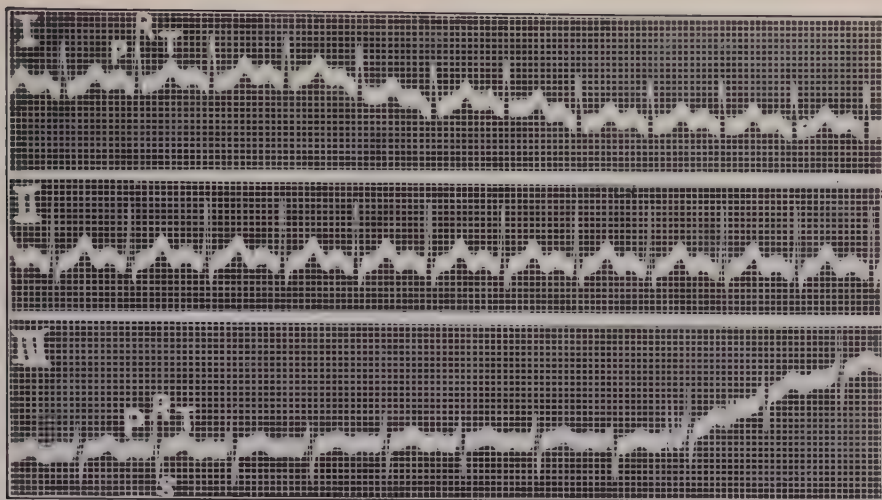


FIG. 84.—CASE 1. F. R.

A case of aortic disease showing the high R in lead 1 and deep S in lead 3 of left axis deviation. Conduction is not delayed. The T in lead 2 is of good amplitude. R_2 is small and notched, the latter a frequent finding in a child's record. Simple tachycardia. Ventricular rate 122.

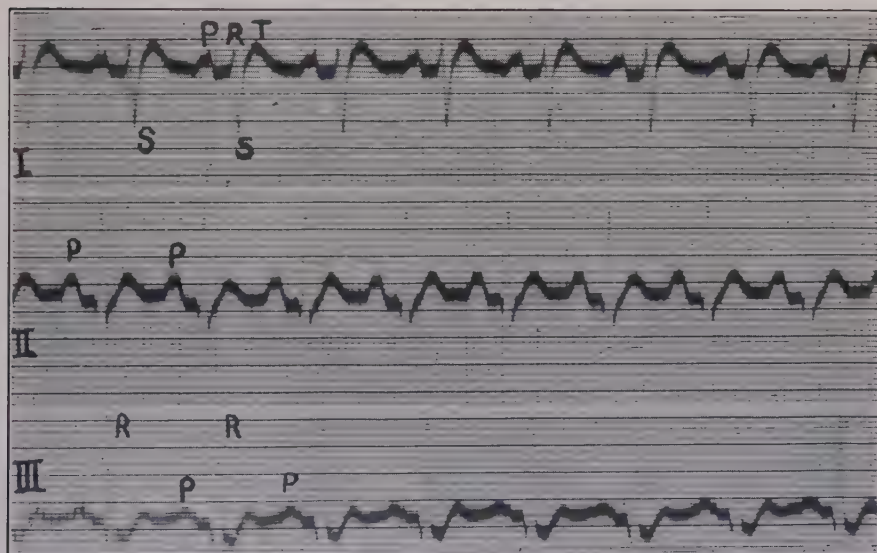


FIG. 85.—CASE 2. L. C.

Preponderance of the right ventricle and notching of the auricular (P) deflections, both findings typical of mitral disease. (The sharp dipping of the T-waves and the slightly prolonged conduction time are digitalis effects.)

CASE 1.—F. R. (Fig. 84), a well-developed Italian boy of nine years, was admitted to the wards with the diagnosis of rheumatic fever. He gave a history of having had swollen and painful joints two years before. His heart was enlarged to the right and left and showed well-developed aortic and mitral lesions. The electrocardiogram recorded a left axis deviation.

CASE 2.—L. C. (Fig. 85), a white girl of seven years, was admitted to the wards with the diagnosis of acute carditis. She gave a history of having had rheumatic fever two and a half years before and had had frequent joint pains since that time. Her heart was markedly enlarged to the right and left, the point of maximum impulse (P.M.I.) being in the left midaxillary line (M.A.L.). There was a loud systolic murmur heard best at the apex and transmitted to the axilla, and a diastolic murmur within the apex. The electrocardiogram showed the notched auricular deflections and right preponderance of a typical mitral stenosis.

Functional Changes in Heartbeat.—*Sinus arrhythmia* is a common type of irregularity of the heartbeat due to alterations in vagal tone. There is a fairly rhythmical rise and fall of the pulse rate in which the heart takes part as a whole. The pacemaker is normal and the sequence of auricular and ventricular contraction is not affected. The variation in pulse periods is found in the length of diastole.

When the vagal tone is low there is an acceleration of the pulse rate during inspiration when intrathoracic pressure is decreased. Conversely the heart rate slows with the increased intrathoracic pressure of expiration.

Sinus arrhythmia increases in frequency and in intensity in the growing normal child as vagal control is acquired. The child with cardiac disease shows the condition less frequently as the presence of sinus arrhythmia varies inversely with the rapidity of the heart rate.

CASE 3.—F. K. (Fig. 86), a white boy of eleven years, was admitted to Bellevue Hospital with a history of occasional epileptiform seizures. During the examination, which was otherwise negative, a marked cardiac arrhythmia was noted. The blood-pressure was 120/70. The electrocardiogram showed a sinus arrhythmia with pulse period variations averaging 0.44 second. During a phase of forced expiration the pulse rate dropped to 60 per minute and was regular.

Simple tachycardia, or rapid heart rate, is present in infancy and in the very young child whose vagal tone is low. It is physiological

after exercise and emotion and the increased activity is transitory. The child with cardiovascular disease usually has a rapid heart rate over long periods of time.

In simple tachycardia the normal sequence of cardiac events rapidly follows one another with only slight, if any, variations in the pulse periods. The electrocardiogram records normal auricular and ventricular complexes.

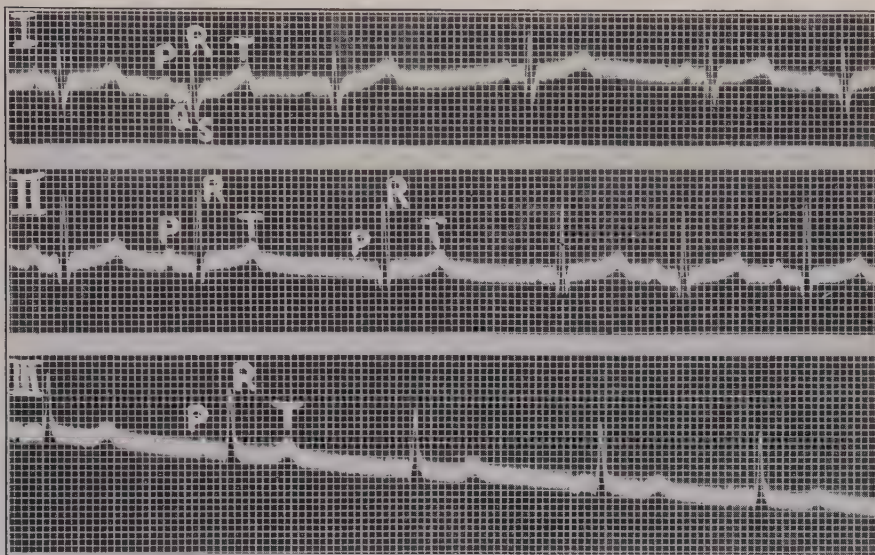


FIG. 86.—CASE 3. F. K.

Leads 1 and 2 show marked sinus arrhythmia with phasic increase and decrease of pulse rate. The greatest pulse period variation is 0.44 second in lead 2. Lead 3 was recorded during an interval of forced expiration and shows a bradycardia with rate of 60 per minute, the effect of vagal stimulation.

CASE 4.—J. T. (Fig. 87, *A*), a white boy of ten years, had been on the wards for several weeks while convalescing from an attack of carditis.

At the time the electrocardiogram was made, he was in good general condition. His temperature was normal, he had no complaints and was "up and about" the ward. His heart, which was well-compensated, showed a typical mitral stenosis and insufficiency with a rate of 90 to 100 a minute.

To test the efficacy of digalen, he was given 30 minims every six hours for twenty-one doses, receiving a total of 630 minims, at which time he showed a bradycardia with very slightly prolonged conduction time (Fig. 87, *B*).

Bradycardia, or slow heart rate, is noted where vagal tone is high. It frequently follows acute infectious disease and is common in the later stage of tuberculous meningitis. It is found in certain brain lesions, especially those affecting the medulla. It is rare in the cardiac child, except in certain cases under digitalis control. Sinus arrhythmia is a usual accompaniment of a slow heart rate. As in a simple tachycardia, the electrocardiogram shows a normal sequence of cardiac events.

Derangement of the Pacemaker and Conducting System.—

Near the opening of the superior vena cava in the right auricle of the heart lies the *sino-auricular node*, the pacemaker of the heart, which is believed by Keith, Flack and others to be derived from the primitive cardiac tube. It contains highly specialized neuromusculature. The muscle-fibers are smaller than those in the rest of the auricle. They are striated and fusiform with elongated nuclei which are in close relation to fine plexuses of nerve-cells and fibers which in turn are connected with vagal and sympathetic nerve trunks.

A stimulus originating in this node passes through the auricular wall by specialized paths, of which we have no definite anatomic knowledge, to the auriculoventricular node, another highly specialized but less sensitive neuromuscular center. From this the stimulus is carried along the ensheathed band of the bundle of His and into its right and left branches. The left division branches freely under the endocardium before giving its ramifications to the two mitral papillary muscles. The right division proceeds directly and subendocardially to the papillary muscles of the right ventricle, a fact that explains the greater frequency of defects of the right bundle. These branches give off final arborizations which become directly continuous with the network of Purkinje fibers which spread through both ventricles and communicate with the ventricular muscle substance proper.

Any inflammation or obstruction which blocks or delays the passage of a stimulus along this path is shown correspondingly in a delay or block in the intervals of the electrocardiographic tracing.

Sino-Auricular Block.—If the natural pacemaker itself be depressed an entire cardiac cycle is affected giving rise to a *sino-auricular block*. The electrocardiogram shows a normal peak or auricular summit with its consorting normal ventricular complex followed by a pause that is nearly equal to twice the preceding pulse period—a lapse in the rhythm during which the sino-auricular node fails to contract and the auriculoventricular node waits for its stimulus. This phase

follows a regular rhythm and usually appears singly or rarely in a tracing, but Lewis has shown that permanent slow action of the heart may be due to this type of inhibition. The depression is frequently found with some degree of auriculoventricular block. Vagus stimulation and poisoning with drugs, as digitalis and quinidin, may cause sino-auricular block.

Sino-auricular block is rare in the child. Wilson says it never occurs. When we remember that vagus control is said to be a fairly late manifestation in childhood, this may be explained as lack of coördinate response to vagal stimulation.

CASE 5.—V. B. (Fig. 87, C) was a poorly nourished boy of eight and a half years, suffering from acute rheumatic fever. The heart-sounds were of poor quality. Mitral stenosis and insufficiency were present. An arrhythmia was noted which on electrocardiographic examination proved to be a block in the sino-auricular node.

Nodal or Atrioventricular Rhythm.—A simultaneous contraction of auricles and ventricles due to an impulse arising in the auriculoventricular node may be caused by depression of the normal pacemaker or by an increase in the activity of the auriculoventricular node. The rhythm is not uncommon in the normal child during the vagal slowing of expiration, or on direct vagal stimulation.

Atrioventricular rhythm is most frequently of irregular degree and an unstable condition. Lewis explains this fact as being due to variations in the level at which impulses arise in the slowly conducting node. He shows the method of its production through the combined stimulation of both the right vagus and left sympathetic.

Atrioventricular or nodal rhythm is recognized in the electrocardiogram by noting the presence of a reduced or absent P-R interval. This may be of opposite sign, that is, the P summits may follow the ventricular deflections which are normal or supraventricular in outline. Both the auricular (P) and the ventricular (Q-R-S) complexes respond to impulses arising from the auriculoventricular node, the P-wave preceding if the level of the impulse formation be high, the Q-R-S if low, and both waves being superimposed if the contractions of both chambers be simultaneous.

CASE 6.—J. B. (Fig. 87, D), a white girl of fifteen years, had been under observation in the Huddleston Memorial Cardiac Class for eight years. Her diagnosis was chronic cardiac valvular disease of rheumatic origin with double mitral and aortic lesions. Classification 2b. Her average pulse rate was 72 to 76 a minute, her blood-pressure

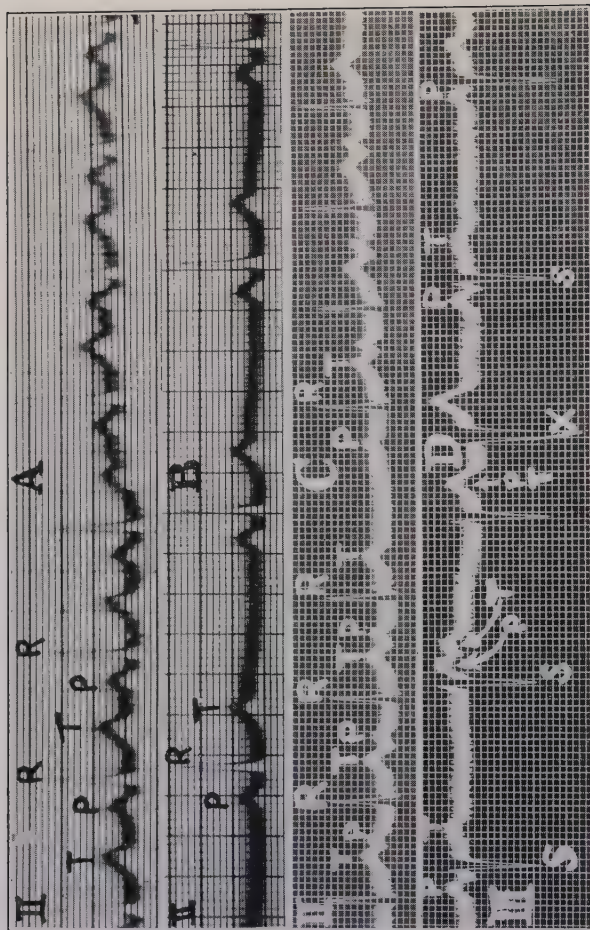


FIG. 87.

A. CASE 4. J. T.—Lead 2. Simple tachycardia with heart rate of 96 per minute. Note the short diastole or rest period averaging 0.12 of a second. The P-R interval is 0.16 second.

B. The same lead in the same person, showing the effect of vagal stimulation under digalen. There is a bradycardia with rate of 52 per minute. The P-R interval is 0.18 second.

C. CASE 5. V. B.—A sino-auricular block is present in the fourth cycle of the lead, where the P-P interval equals 1.14 seconds, almost twice the period of the preceding or succeeding cycles which are 0.60 of a second. There is an impairment of auriculoventricular conduction, the P-R interval being 0.24 second in duration.

D. CASE 6. J. B.—Lead 3. Atrioventricular rhythm of unstable degree. In the second and third complexes the auricular deflection (P) is seen to follow the ventricular contractions which arise from nodal stimulation. The fourth event (X) is a premature contraction arising in the left ventricle.

100/50. Her cardiac reserve was low. She entered the hospital with early congestive failure. She had a short cough. Dyspnea was marked. The liver was tender and slightly enlarged. There was edema of the ankles. The lungs showed congestion at both bases. The temperature was 103° F. and the pulse rate 120. She was digitalized and became nauseated. Her pulse was slow and regular. The electrocardiogram showed nodal or atrioventricular rhythm.

Heart-Block, Partial and Complete.—Heart-block is the term given to the condition of obstructed stimulus transmission between the upper and lower chambers of the heart. The degree of resistance met by the stimulus may vary from partial to complete blockage in the conducting pathway. However great the delay, if there be ventricular response to auricular stimulation the block is only partial (Fig. 88, *A, B, C*). When, on the other hand, the dissociation is complete between the upper and lower chambers of the heart, the ventricle, no longer able to conform to auricular control, finds its new pacemaker in the auriculoventricular node or in the junctional tissue and contracts usually in a slow regular rhythm. At the same time the auricle continues a more rapid rate of contraction in response to the sino-auricular node. Although there is no definite relationship between the two rhythms in complete heart-block, Lewis has shown that the auricular impulse following a ventricular contraction is slightly hastened (Fig. 88, *D*).

Conduction may be retarded or obstructed by any break in the integrity of the transmission pathway through the auriculoventricular node, the bundle or its branches. Such may be caused by the invasion of heart muscle through acute or chronic infection, lues, malignant growths, anatomical malformation of the tracts, vagal stimulation and by certain toxins in the blood stream, such as found in asphyxia or in overdosing with certain drugs, as digitalis and quinidin.

In the child, partial or complete block is not uncommon in acute rheumatic carditis and acute infections, in asphyxia and in congenital lues. Complete heart-block is less common than a partial dissociation. The reported cases have usually been due to congenital defect in arrangement of the conducting system, to overdosing with digitalis, to diphtheria, poliomyelitis, influenza or asphyxiation.

As a temporary finding, derangement in the conduction is probably preceded by toxic or inflammatory changes in the transmission system. When the condition is constant there is chronic myocardial damage and is a basis for very poor prognosis except where the lesion is due

to a congenital defect in the conducting system and not to any tissue degeneration.

In the electrocardiogram an impulse leaving the sino-auricular node and meeting with resistance in its passage to the ventricles gives a definite picture. There is a lengthened (Fig. 88, *B*) or absolutely irregular P-R interval (Fig. 88, *D*) according to the degree of block encountered. Although the average duration of the P-R interval in a child is 0.12 second, conduction is not considered delayed if less than 0.2 second be consumed in the passage of the stimulus from the auricle to the ventricle through the bundle, its branches and arborizations.

CASE 7.—W. McS. (Fig. 88, *A*), a frail white boy of nine years, was admitted to the wards suffering from acute rheumatic fever and carditis. His physical condition was poor. He was undernourished and anemic. His temperature was elevated and his pulse rapid. His eyes were bright and his skin was drawn. Many subcutaneous rheumatic nodules were found over the tendons of his fingers, wrists, elbow-joints and knees. His heart was enlarged to the left and a double mitral lesion was present. A pericardial friction-sound was audible. He had had digitalis. The electrocardiogram showed a progressive type of partial block evidently due to degenerative changes affecting conduction. The child died two days later. No autopsy was performed.

CASE 8.—B. S. (Fig. 88, *B*), a poorly nourished white girl of eleven years, was admitted to the wards of Bellevue Hospital suffering from cardiac failure of rheumatic origin. Her general condition was poor. There was marked orthopnea and mental distress. The abdomen was distended, the liver enlarged and tender. There was no edema of the extremities. The heart was markedly enlarged and over-acting. A loud presystolic murmur was audible over the apical region. A systolic murmur was present over the same region and transmitted upwards and to the axilla.

Figure 88, *B* is from a tracing taken during convalescence when the patient had for several weeks been on a maintenance dose of digitalis—in her case, 0.05 gram twice daily. The record shows a marked delay in the conduction between the auricles and the ventricles which was transitory and entirely a digitalis effect, disappearing when the drug was discontinued.

CASE 9.—G. C. (Fig. 88, *C*), a male infant of two and a half years, was under observation at Bellevue infants' clinic for eighteen months suffering from congenital heart disease and malnutrition. The child developed poorly. He sat up in bed but could not walk. There was

slight cyanosis of the lips and skin. The heart was large and globular in outline. There was a loud, blowing, systolic murmur over the precordium transmitted over the entire chest. The electrocardiogram varied on different occasions, but always showed some degree of obstruction which was probably caused by a mechanical interference in the pathway of conduction. The tracing recorded a 2:1 block.

CASE 10.—M. T. (Fig. 88, D), a boy of eleven years, reëntered the wards with acute rheumatic fever. He had had several previous attacks.

He was in great distress with dyspnea and palpitation. He complained of a sensation of dizziness. Several joints were acutely swollen.

On examination, signs of cardiac failure were noted. The lungs were congested, the extremities edematous. The heart showed impulse over the entire precordium. The point of maximum impulse was felt 10 centimeters to the left of the midsternal line. The sounds were obscured by murmurs. The pulmonic second and aortic second sounds were split. A diastolic blow was present along the left sternal border. A short presystolic and loud systolic were heard at the apex, the latter transmitted to the axilla. The pulse was irregular. The blood-pressure was 98/48. The x-ray showed the heart enlarged to right and left with accentuation of the left auricular curve. Full doses of digitalis were given. An electrocardiogram recorded complete block.

Bundle-Branch Block or Defect, and Arborization Block.—A bundle-branch defect is the change in conduction caused by an obstruction in one of the main divisions of the bundle.

The condition is not uncommon in lues and aortic disease and has been noted as a temporary finding in acute carditis where it appears to be of serious import. No case has so far been reported in a young child.

The electrocardiogram is typical (Fig. 89). There is a widening of the Q-R-S interval above its normal limit of 0.1 second, a notching of the same complex, and the following T-wave is in the opposite direction of the main deflection. Another distinguishing feature is that of the increased amplitude of the ventricular deflections. These peculiarities may be explained by the unilateral course taken by the stimulus, unbalanced at first by that in the opposite ventricle. The picture shows a levo- or dextrocardiogram according to the bundle affected, a right bundle-branch defect giving left predominance and vice versa.

A block in a portion of the main branch, usually the left one, or in the final arborizations of the system has been shown by Op-

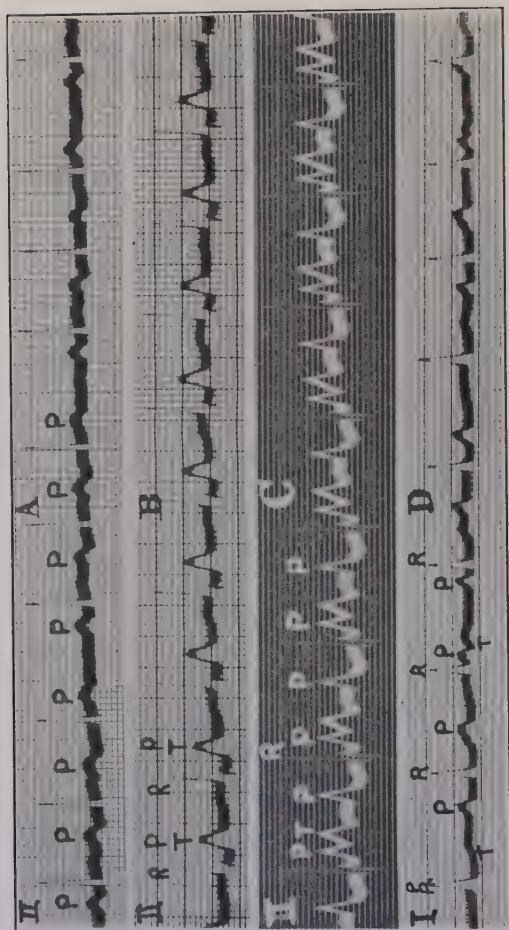


FIG. 88.

A. CASE 7. W. McS.—Lead 2. Progressive partial block. If the above P-R intervals be measured successively it will be noted that there is an increase in the P-R interval until there is an auricular complex to which the ventricle fails to respond; the ventricular refractive period is not over until the next auricular contraction has taken place.

B. CASE 8. B. S.—Lead 2. Partial block in which the P-wave is superimposed on the T-deflection, the P-R interval being 0.38 second. (See also Fig. 87.)

C. CASE 9. G. G.—Lead 2. Partial block of the 2:1 type, in which the ventricle responds to every second auricular contraction.

D. CASE 10. M. T.—Lead 1. Complete block. The auricles are contracting regularly at the rate of 132 per minute, the ventricles at 92 per minute.

penheimer to differ in its tracing from that of a main lesion by having ventricular deflections of low amplitude.

CASE II.—A. D. (Fig. 89), seventeen years of age, had attended the clinic since her seventh year when she had chorea. At twelve years of age she had acute rheumatic fever. She now complained of palpitation. The patient was tall and thin and had little cardiac reserve. Some purpuric spots were found scattered over the body and extrem-

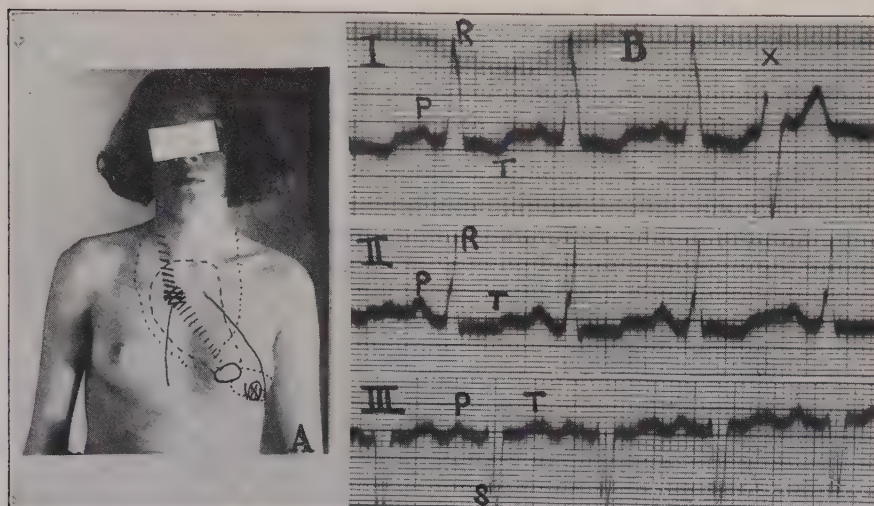


FIG. 89.—CASE II. A. D.

A. The patient, showing areas of aortic and mitral murmurs diagrammatically by means of dotted lines for systolic and dashes for diastolic murmurs. The presystolic rumble is outlined with an unbroken line.

B. Electrocardiogram shows an early defect in the right branch of the bundle. The picture is a levocardiogram. The ventricular complexes are of great amplitude, they are notched or split. The T-waves are in the opposite direction of the main deflection. There is an irregular widening of the Q-R-S interval. The P-R interval is 0.2 second.

The fourth ventricular complex in lead I is a premature contraction arising in the right ventricle.

ities. The blood-pressure was 64/0. The pulse rate of 120 per minute was interrupted by frequent premature contractions. The heart was enlarged, the point of maximum impulse being 14 centimeters from the midsternal line in the sixth left interspace. A systolic thrill was present over the second right interspace and a diastolic thrill over the apex. Systolic and diastolic murmurs were audible over the aortic area and a loud systolic blow and presystolic rumble were heard at the apex.

The child refused to enter the hospital and died a few weeks later at her home.

The electrocardiogram showed a defect in the right branch of the bundle with frequent premature contractions arising in the right ventricle.

Auricular Fibrillation.—Auricular fibrillation is the most severe disorder of cardiac rhythm compatible with life. The auricles being in a state of continuous tremulous movement, systole is not accomplished and ventricular response is completely irregular in rate and quality.

The crippled heart of rheumatism is most prone to fibrillation. The condition is rare in the child, and when it occurs is of serious prognostic import. It is commonly noted that vagal stimulation will cause fibrillation in the toxic or predisposed heart, thus in acute endocarditis where digitalis has been forced this arrhythmia occasionally results.

The electrocardiogram in auricular fibrillation is characteristic. There is an absence of normal auricular contractions or P-waves. They are replaced by oscillations which are not uniform in rate, amplitude or form. The Q-R-S or ventricular complexes are supraventricular in type, they fall in irregular sequence and their amplitude is frequently varied.

CASE 12.—R. H. (Fig. 90), a colored boy of nine years, had been on the wards of Bellevue Hospital for some weeks suffering from acute carditis and congestive failure, rheumatic in origin.

The boy's condition was poor. He was orthopneic and complained of severe pain in the precordium. There was marked abdominal distention and edema of the extremities. The blood-pressure was 130/80. The heart was greatly enlarged to the left. The tricuspid as well as the mitral valves were affected. Digitalis was being given in full doses. The pulse rate had become irregular on the day the electrocardiogram was taken.

Ventricular Fibrillation.—This is almost always a terminal event and since it is of short duration is rarely noted. As in the auricular type the condition may be initiated in vagal stimulation. Premature contractions and paroxysmal tachycardia are the usual precursors. The electrocardiogram records a series of totally haphazard waves which are irregular in size and form, falling without response to any guiding stimulus. There is an anarchy in the fibrils of the ventricle.

Auricular flutter is a tachycardia induced by rapid and regular auricular contractions with a partially blocked ventricular response. The mechanism of the action is theorized by Lewis as being due to the stimuli passing over the auricular muscle in circus movement

so that the waves are contiguous to one another and some group of muscle-fibers is constantly in contraction. Flutter is rarely, if ever, seen in the child. The electrocardiogram shows a stream of regular P-waves which are intercepted every second or third beat by ventricular complexes of supraventricular type. The As-Vs block may be of higher degree or may be of mixed type (Fig. 91, A).

Paroxysmal tachycardia is a type of rapid and regular heart action,

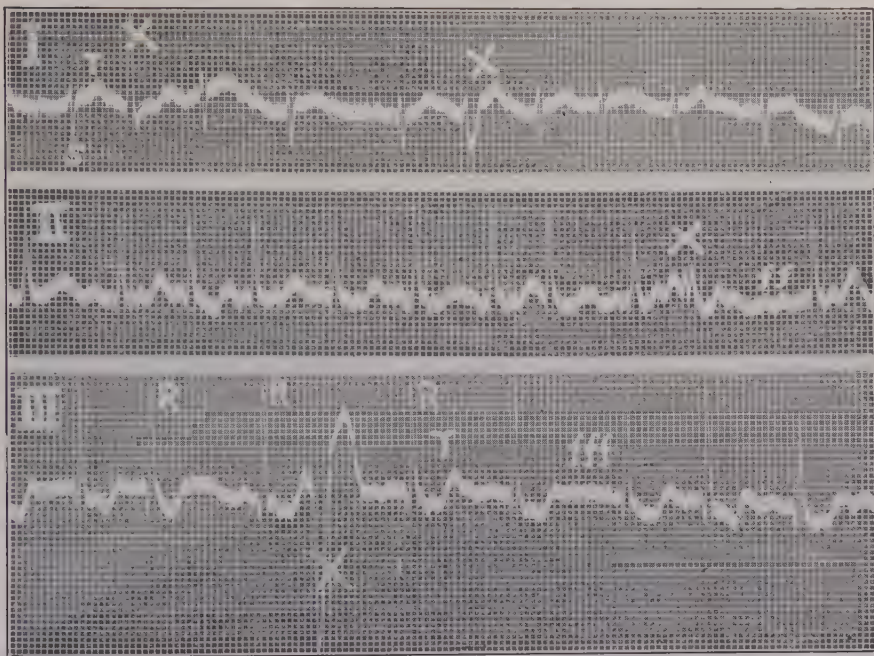


FIG. 90.—CASE 12. R. H.

Three leads. Rapid auricular fibrillation with coarse auricular oscillations and right ventricular preponderance. In lead 1 a left ventricular premature contraction is the second ventricular event, the sixth is one the origin of which is in the right ventricle. In the third lead the fourth ventricular contraction is a premature one arising in the left ventricle.

the beats originating in abnormal or "ectopic" foci. The new focus may be in the auricle (see Fig. 91, C), the auriculoventricular node, the junctional tissues or in the ventricle. If the myocardium is damaged it may be produced as an effect of vagal overstimulation. The condition is, however, preceded and followed by frequent premature contractions. The prognosis is poor in the child where it is uncommon.

The electrocardiogram shows a tracing of regular abnormal beats

following one another in rapid succession. It is occasionally impossible to determine the site of the ectopic focus unless the beginning or the ending of the paroxysm is seen.

CASE 13.—S. P. (Fig. 91, *B*), a Jewish boy of ten and a half years, had attended the cardiac clinic following a first attack of chorea three years before. His mother and maternal grandfather had Huntington's chorea.

The boy had infected tonsils which caused him frequent trouble. Their removal was refused. Attacks of chorea recurred regularly.

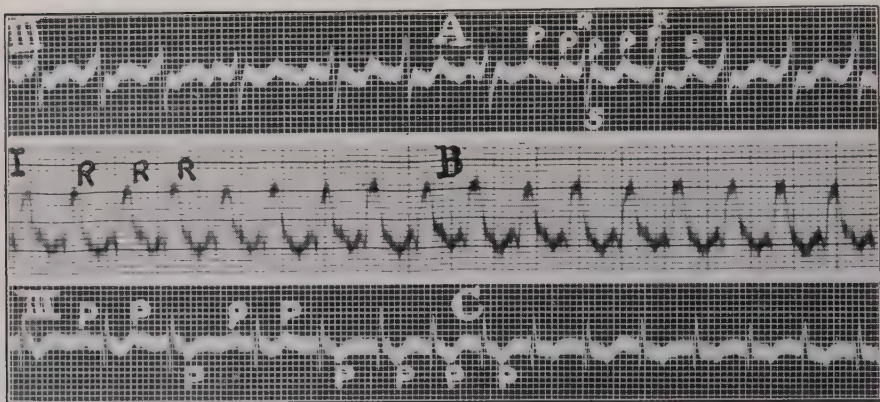


FIG. 91.

A. R. G.—Auricular flutter recorded in a young Jewish woman of twenty years. The regular, wavy curve of the auricular (P) deflections are interrupted every second or third beat by ventricular (R) complexes.

B. CASE 13. S. P.—Lead 1 showing paroxysmal tachycardia of the ventricular origin. Rate 255 per minute. The Q-R-S-T complex resembles a ventricular premature contraction, the interval is widened and notched and the T in the opposite direction to the main deflection.

C. T. McC.—Lead 3 showing a 1:1, 2:1 partial block preceding paroxysm of tachycardia arising in the auricle. Note the inversion of the P deflections.

The boy first complained of a "nervous spell" after climbing a flight of stairs. These attacks, which later proved to be paroxysmal tachycardia, were accompanied with apprehension and occurred frequently. They usually lasted four to six hours, were relieved by vomiting and were followed by profuse perspiration.

The point of maximum impulse was 9 centimeters to the left of the midsternal line in the fifth interspace. The pulmonic second sound was accentuated. Loud systolic and diastolic murmurs were present at the apex. Electrocardiographic tracings showed paroxysmal tachycardia, ventricular in type.

dren only one premature contraction was noted and that only after the records had been repeated in many instances over a series of years.

The Congenital Heart Defect.—In the congenital heart the electrocardiogram occasionally aids in the diagnosis of the lesion present. Typical pictures are given by the transposed heart and that in which a stenosis of the pulmonic valves is present. In the pure dextrocardia (Fig. 92) all the summits in lead I are inverted and leads 2 and 3 replace one another. In pulmonic stenosis (Fig. 93) there is a marked



FIG. 93.—CASE 15. H. P.

A record of pulmonic stenosis. The ventricular deflections are of great amplitude suggesting a congenital heart lesion. Right ventricular preponderance is marked, the S in lead I being 17 mm. deep and the R in lead 3 being 22 mm. high.

right preponderance. No changes are characteristic for septal defects or patent ductus arteriosus.

CASE 14.—P. M. (Fig. 92), a white boy of five years, was admitted to the ward with the diagnosis of possible typhoid fever. During the examination it was noted that the heart lay in the right side of the chest; the apex-beat was palpable in the fourth right interspace 5.5 centimeters from the midsternal line. A systolic blow was heard over the precordium. The pulmonic second sound was present. The electrocardiogram showed dextrocardia with low voltage.

CASE 15.—H. P. (Fig. 93), a mentally deficient, twelve-year-old white boy attending the cardiac clinic, showed a poor general condi-

tion. His physical as well as mental development was retarded. There was some cyanosis of the extremities with clubbing of the fingers. The heart was enlarged to the right and a tearing systolic murmur was present over the entire heart, being loudest over the pulmonic area. The second pulmonic sound was absent. The electrocardiographic diagnosis was pulmonic stenosis.

CHAPTER XII

THE HEART IN OTHER INFECTIONS

SUBACUTE BACTERIAL ENDOCARDITIS

This term is applied to the type of endocarditis which is caused by *Streptococcus viridans* or other anhemolytic streptococci. It runs a subacute course over a period of months. Malignant endocarditis is a term used to describe any endocarditis which runs an acute, rapid course resulting in death, but the subacute form is malignant, since the result is almost invariably the same. Any bacterial endocarditis may become ulcerative. Verrucous endocarditis refers to an endocarditis with vegetations, and does not differentiate clinical entities. Subacute bacterial endocarditis (or endocarditis lenta) particularly is to be differentiated from rheumatic carditis. This is sometimes difficult clinically, especially when the patient is in the phase where the blood-culture is sterile. That the disease is more common in children than was formerly realized has recently been pointed out by Rost and Fischer, although only about one hundred cases in children have been reported in the literature.

Rheumatic fever definitely predisposes to subacute bacterial endocarditis, and in a certain small percentage of cases, congenital malformation of the heart seems to be the predisposing factor. In other words, valves which have previously been diseased form the basis for this form of endocarditis. The relation of endocarditis lenta to rheumatic fever is close, both clinically and bacteriologically, but at present the situation is still confused. It has already been stated that patients with rheumatic fever give strong skin reactions to filtrates of streptococci. In subacute bacterial endocarditis, on the other hand, the patients give no skin reaction. On animals it has been shown that endocarditis can be produced only if the animal has previously been partially immunized. It seems paradoxical, but apparently true, that in subacute bacterial endocarditis, there is an active and almost always fatal infection in an individual who is biologically immune.

How the bacteria which colonize on the valves reach the valve is



FIG. 94.—HEART OF YOUNG ADULT WHO DIED OF SUBACUTE BACTERIAL ENDOCARDITIS ON CONGENITAL MALFORMATION OF HEART.

Ulcerative and vegetative endocarditis of aortic valve and of defective interventricular septum. Vegetations also on mitral valve and wall of left auricle. (Bellevue Hospital Pathological Museum, Collection of C. de la Chapelle.)

not clear. Possibly a mass of bacteria circulating in the blood stream are caught on the valve, and because the factors of resistance of the valve are low, the growth of a colony follows. If the colony becomes large enough, some of the bacteria are so deep in the tissue that no bactericidal agent can reach them. If this be true, it would explain why no therapeutic measures are of avail.

The vegetations are much more friable than those of rheumatic fever, break off easily and enter the blood stream, causing the emboli

which are a prominent feature of the disease. Vegetations may occur on the auricular walls, the chordæ tendineæ and ventricular walls, as well as on the valves (Fig. 94).

The patient with subacute bacterial endocarditis appears chronically ill, has a yellowish pallor to the skin and frequently cyanosis and some clubbing of the fingers and toes. The symptoms of a low-grade toxemia are present, with lassitude, poor appetite, feverishness, and pains in the extremities. The onset may be acute, with chills and a high fever.

An eleven-year-old boy at Bellevue presented a fairly typical case (Fig. 95). At nine years of age he had an attack of rheumatic polyarthrititis, but had no knowledge then of any involve-

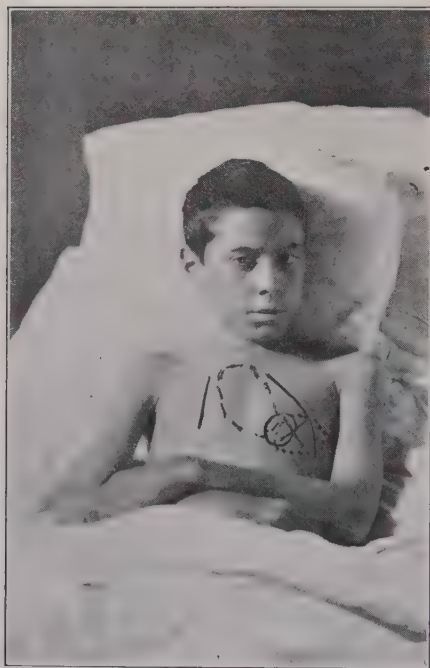


FIG. 95.—SUBACUTE BACTERIAL
ENDOCARDITIS.

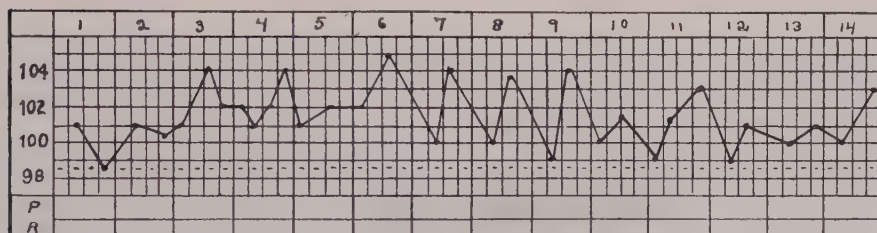
Described in text. Note comparatively good general condition.

ment of the heart. About a month before admission to the hospital he began to feel sick, with malaise, loss of appetite and headache, but no localized symptoms. He was taken to a physician who diagnosed heart disease and referred him to the hospital. He had fever, leukocytosis (23,000), dyspnea and pallor. Physical examination revealed an enlarged heart with signs of mitral stenosis and insufficiency and aortic insufficiency. He also had an aortic systolic murmur without other signs of aortic stenosis. This may have been caused by an aortitis. Repeated

blood-cultures showed *Streptococcus viridans*. He had several crops of small petechiæ on the extremities, but none in the conjunctivæ or eye-grounds. There was gradual wasting, and the *café au lait* pallor became more marked. Besides the petechiæ, there were groups of pink spots on the feet and palms of the hands, which were 2 to 5 millimeters in diameter, not raised, and which faded on pressure. In the palm of the right hand there occurred a tender nodule, over which the skin was slightly reddened (Osler's node). This persisted for about ten days. The boy had pain in the calves of the legs, and in the knee-joints and ankle-joints. There was no change in the heart size, but the mitral murmurs increased in intensity. His fever persisted.

CHART IV

TEMPERATURE CURVE IN SUBACUTE BACTERIAL ENDOCARDITIS



White blood-count
12,000
Polys 80 per cent
Hemoglobin 45 per cent

21,000

Male, aged 11 years

Repeated blood-cultures positive: *Streptococcus viridans*

Diagnosis

1. Rheumatic heart
 - Mitral stenosis and insufficiency
 - Aortic insufficiency and roughening
2. Subacute bacterial endocarditis

The leukocyte count varied between 15,000 and 20,000, with increase in the polymorphonuclear cells. The course with this child was progressively downward over a period of months. The diagnosis was subacute bacterial endocarditis (*Streptococcus viridans*) superimposed on a heart already damaged by rheumatic fever.

Although his spleen was not palpable there was definite tenderness over the splenic region. He showed no signs of pericarditis, which is common in acute rheumatic infection of the heart, and no rheumatic nodules. He had an embolus in the left popliteal artery and a few days later an embolus lodged in the left brachial artery. The second embolus was followed by an aneurysm of the brachial artery just above the site of the embolus.

The diagnosis of subacute bacterial endocarditis in any individual rests on the finding of petechiæ, enlargement of the spleen or tenderness in the left hypochondrium, pallor, wasting, fever, clubbing of the toes and fingers and embolic phenomena. Tender nodules on the pads of the hands, fingers and toes (Osler's nodes) are pathognomonic, and sometimes there is tenderness over the tip of the sternum. Red blood-cells are frequently found in the urine. A positive blood-culture is a great help in making the diagnosis, but it must be remembered that there may be bacteria-free periods during the course of the disease.

A few cases of recovery have been reported, but the prognosis is very bad. The course runs from four months to two and a half years. Death is frequently caused by cerebral embolus, although intercurrent infection or failure of the heart or kidneys may be the immediate cause of death.

No form of therapy has so far afforded more than temporary relief.

THE HEART IN ACUTE ILLNESS

In any acute infection, the condition of the heart is the chief factor on which is based the probability of recovery, death or partial recovery. Certain infections are more apt to include some involvement of the heart, either from the effect of the toxins on the myocardium, or actual invasion of the heart by the bacteria causing the disease.

Pneumonia.—In pneumonia, death is usually from heart-failure, rarely induced by the sudden strain brought on by the crisis. Pneumonia, as we see it in children over two years, is a less severe disease than in either adults or infants. In 116 cases of lobar pneumonia in children over two years of age, transient systolic murmurs heard at the apex or at the pulmonic area were noted in only nine cases. In one case premature contractions were found, and in one the heart was displaced to the left and there was a loud, rough, blowing systolic murmur along the left border of the sternum. No autopsy was performed on this case. In seventy-six children under two years with lobar pneumonia, only one had a transient systolic blow. Of fifty-four infants with bronchopneumonia there were no special heart signs, except that at the end the heart became very rapid with feeble heart-sounds. Since it is felt that in lobar pneumonia in children, both under and over two years of age, the prognosis is good, and the heart is seldom put to too great a strain, no particular measures, other than nursing care, are used as a routine to spare the heart. In severe cases

with signs of overburdening of the heart, digitalis is used as a supporting measure. The children with bronchopneumonia, however, are given digitalis as a routine measure. How great a help this is cannot be stated at present, but the rapid, poor heart action, and the poor prognosis, indicate the need for a cardiac stimulant.

Occasionally in pneumonia, pericarditis develops, and it is probably more common than is recognized clinically. Vegetations occur, and constitute a real malignant endocarditis. Osler found that of 209 cases of malignant endocarditis fifty-four occurred in pneumonia. About 10 to 15 per cent show pericarditis at autopsy, while only 1.5 to 2.5 per cent are recognized clinically.

The presence of a systolic murmur probably indicates a relaxation of the heart muscle rather than a definite lesion. Endocarditis is indicated by a diastolic murmur. Pericardial friction-rub should be looked for but is often difficult to recognize because of the intrapulmonary signs or a pleuropericardial rub. The pericarditis is usually fibrinous, although there is sometimes a small collection of fluid.

The heart rate is increased during the acute stage of the disease, and is often followed by a bradycardia. Any type of arrhythmia may occur. Dilatation of the heart should be watched for and the quality of the second pulmonic sound observed, since weakening of this sound may forecast myocardial insufficiency.

Regarding the treatment of the heart in pneumonia, many feel that digitalis should be given as a routine in all cases in the hope of avoiding sudden heart-failure. Caffein or adrenalin by hypodermic injection may be resorted to in an emergency, but, as Swift states, "when in any infection, be it general or cardiac, the heart muscle and conduction system are too badly damaged to respond to digitalis, the patient is, indeed, in a precarious condition."

Diphtheria.—The heart is affected to some extent in most cases of diphtheria. Evidence of this may be simply a rapid and feeble heart action, but irregularities in the rhythm are common. Premature contractions occur frequently; nodal rhythm, heart-block and even auricular fibrillation have been noted. These arrhythmias are readily demonstrated by the electrocardiograph.

During the acute stage there may be a systolic murmur, but this may be febrile and not indicative of any actual effect on the heart by the toxin of diphtheria. Symptoms from the heart occur usually in the second or third week of the disease. There may be a sudden drop in the heart rate, or very rapid action with gallop rhythm, epigastric

pain and tenderness. Because of the myocardial degeneration, sudden fatal dilatation of the heart may occur. During convalescence and sometimes for months after the attack of diphtheria, periods of paroxysmal tachycardia may occur. Usually there is no permanent damage so that functionally recovery is complete.

The pathologic lesion consists of patchy degeneration of the muscle-fibers of the heart. Edema, collections of wandering cells and proliferation of fixed connective-tissue elements follow in the interstitial tissue. Since the degeneration occurs in patches, enough undamaged tissue may be left to allow the reparative process to proceed in the affected areas. The final outcome depends on the ratio between the amount of damaged and undamaged myocardium.

Scarlet Fever.—During the early acute stage of scarlet fever, the same type of toxic cardiac symptoms may occur that supervene in diphtheria. Later in the disease, the prolonged action of the infecting agent of scarlet fever sometimes produces a carditis comparable to that found in rheumatic fever. Polyarthritides not infrequently is one of the complications or sequelæ of scarlet fever. That the connection between scarlet fever and rheumatic fever is fairly close seems to be indicated by the histories of some cases. An attack of polyarthritides with carditis may follow scarlet fever. In 10.2 per cent of Wilson's cases of rheumatic heart disease, there was a history of scarlet fever, of which 54.3 per cent had preceded the carditis and 15.4 per cent occurred in the same year as the first manifestation of rheumatic fever. The age incidence of scarlet fever coincides with that for rheumatic fever. Since the etiological agent in scarlet fever is known to be a streptococcus and is believed to be a streptococcus in rheumatic fever, this connection between the two diseases is probably more than a coincidence.

The symptoms and physical signs of heart involvement in scarlet fever are the same as those of rheumatic carditis and therefore need not be described here. The changes found in the heart at autopsy are identical or very similar to those found in rheumatic carditis.

Typhoid.—Usually, the heart-sounds during an attack of typhoid fever remain entirely normal, although in very severe cases a soft systolic blow along the left margin of the sternum may be heard. Gallop sound is fairly common. In the severe cases the heart-sounds become feeble and the first and second heart-sounds become nearly alike (embryocardia). Pericarditis and endocarditis are both rare. Myocarditis occurs more frequently and is indicated clinically by the

weakening of the heart-sounds and poor functional action of the heart.

Septicemia.—Septicemia, whether it is caused by the staphylococcus, streptococcus, pneumococcus or other microörganism, usually involves the heart. The lesion is an endocarditis, with vegetations on and ulcerations of the endocardium of the valves. The heart muscle becomes weakened as a result of the severe intoxication and cachexia. The chief danger and importance of the attack on the endocardium lies in the frequency with which infective emboli are swept into the blood stream, carrying the bacteria to other parts of the body. The physical signs do not differ from those of any other form of endocarditis. Death occurs in most cases before the mechanical factors following inefficient heart valves become prominent features of the disease.

Nephritis.—Acute hemorrhagic nephritis as it is seen in children is considered to be an infection. Usually there are no signs in the heart, but slight enlargement, gallop sound, a systolic mitral murmur or accentuation of the aortic second sound may occur. Sudden dilatation of the heart occasionally supervenes, with dyspnea and pulmonary edema. Premature contractions are noted in some cases, but are considered to be toxic in origin.

Hypertrophy of the heart is a frequent finding in chronic nephritis, and is thought to be secondary to the vascular changes which put extra work on the ventricle. The physical signs are no different from those of hypertrophy from any other cause.

Treatment directed toward the heart itself is seldom necessary, although digitalis, which acts directly on the heart, may be of value in some cases.

CHAPTER XIII

DIAGNOSIS AND CLASSIFICATION OF HEART DISEASE

DIAGNOSIS

The diagnosis in a case of heart disease, or of suspected heart disease, is complete only after the etiology, the anatomical changes, the physiological condition and the functional capacity have been studied and carefully analyzed. In the Appendix will be found a system of nomenclature to be used in diagnosis which has been approved by the American Heart Association. It is an outline of the points to be considered in making a diagnosis, and a study of this will show what a complete diagnosis really involves.

Etiological.—In children the etiological diagnosis depends partly on the anatomical, since not infrequently the decision has to be made between acquired and congenital lesions. The history should be taken carefully before the physical examination is made, and it will often provide a hint which will help in the final diagnosis. If there is a definite history of rheumatic fever, or if the examination reveals that the condition is a congenital one, the etiological diagnosis is clear. There are many cases of heart disease with no history of rheumatic fever, but with an anatomical condition which is clearly rheumatic in origin. For instance, mitral stenosis is always a rheumatic lesion, just as *tabes dorsalis* is always considered syphilitic even in the absence of a history of a primary lesion. A murmur of mitral insufficiency with enlargement of the heart can be assumed to be rheumatic in the large majority of cases. Aortic disease is also usually rheumatic in children. If there is abnormal cardiac function, with no history pointing to the cause, the etiological diagnosis is "unknown."

Certain cases are very puzzling, particularly those with definite congenital lesions that also give a history of rheumatic fever. The etiology is clear enough, but the structural changes may be impossible to differentiate exactly.

When a child is seen in an attack characteristic of rheumatic carditis (previously described), the etiology is certainly rheumatic, even though there is no arthritis or chorea, or any history of such ill-

nesses. The possible etiological association of rheumatic heart disease with scarlet fever and occasionally other infections, must not be overlooked. In making the etiological diagnosis it is most important to state whether the infecting agent is active or inactive.

Anatomical.—In estimating the condition of a patient it is logical to take up the anatomical diagnosis before the functional, since treatment depends on whether or not heart disease is actually present, and if present, how extensive the lesions are. The complete diagnosis cannot be made until the type of lesion is determined. In the chapter on examination of the heart we have discussed the method of study in logical sequence. The examination first determines the *size* and *action* of the heart and this tells us much about the heart muscle. This is, after all, the main factor in determining the extent of the damage to the power plant. A heart which is not much enlarged and whose action is slow, regular and forcible is probably not seriously damaged, at least in its function.

The differentiation of the various murmurs is based on the characteristics which have already been described in Chapter VIII, but certain points will be emphasized here.

Systolic murmurs are most apt to cause inaccurate diagnosis. All the characteristics of the murmur need consideration and should be systematically studied. The time in the cycle, total area, maximum site and maximum transmission are the most important facts to be noted. The warning to time the murmur only by a finger on the apex cannot be too often repeated. The total area of audibility of the murmur should be studied so that the maximum site and transmission will be accurately located. The pitch, quality and intensity are less important, but should be noted.

The murmurs found on examination are the chief evidence pointing to *disease of the valves*. The presence of a low-pitched rumbling diastolic murmur at the apex, sometimes with presystolic crescendo, is characteristic of *fibrosis of the mitral valve* (*i.e.*, mitral stenosis). The first sound is short and snapping. In corroboration, the x-ray shows that the left auricle is enlarged. These three points give adequate proof of mitral stenosis. The only important differential condition is the diastolic murmur which occurs at the apex in cases of aortic insufficiency (Austin Flint murmur), in the absence of disease of the mitral valve. If the aortic insufficiency is rheumatic in origin, the murmur is probably caused by mitral stenosis. If there is a systolic murmur, the presystolic is probably caused by mitral stenosis. In the

absence of a mitral systolic murmur, it is impossible to differentiate the Flint murmur from that of a mitral stenosis.

Mitral insufficiency is probably too often diagnosed when it is not present; but a mild carditis often remains undetected. The minimum evidence on which the diagnosis of insufficiency should be made is (1) the presence of a constant systolic murmur, heard best at the

apex or just outside the apex, usually transmitted chiefly toward the axilla, and either following or replacing the first sound; and (2) enlargement of the heart. The enlargement may be so slight that it can only be determined with certainty by the x-ray.

Other systolic murmurs are functional, febrile, those caused by aortic stenosis or roughening, septal defect, pulmonic stenosis, cardiorespiratory, and the pericardial and pleuropericardial rubs.

Functional murmurs are of several types:

1. Soft systolic blows heard loudest at the apex and not transmitted to the left of the apex (Fig. 96). The total area may be small, a few centimeters in diameter, or may be heard over most of the precordium. Such a murmur is particularly to be distinguished from that heard in an



FIG. 96.—NO CARDIAC SYMPTOMS.

Heart (R. M.)

June, 1920. *Size, action and sounds* normal. *Murmur*: Musical systolic, short; inside apex not transmitted.

December, 1921. *Murmur*: Faint, systolic, blowing, not musical, heard only when patient holds breath.

May, 1922. *Murmur*: Hardly audible.

acute carditis. If the murmur is functional, the signs of infection (slight fever, leukocytosis, pallor, fatigue, etc.) will be absent.

2. Similar systolic murmurs heard loudest at the pulmonic and surrounding areas (Fig. 97). With these two murmurs there is no cardiac enlargement, and no accentuation of the second sound. The murmurs may change with posture, disappearing or coming out only on lying down. They are usually heard in pale, undernourished children

and in later childhood. Various explanations have been offered to account for the mechanism, but none are entirely satisfactory. They have been called hemic because they are often seen in anemic children, but are sometimes not found in very anemic individuals. They may perhaps be due to relaxed orifices in a child who is generally flabby.

3. Febrile murmurs are common especially when the temperature is high. They are usually systolic, apical and not transmitted. They may be heard over any part of the heart and are soft in quality rather than harsh. They usually disappear promptly with the cessation of fever and slowing of the pulse.

4. "Musical functional" murmurs. These are short, musical or grunting systolic, or late systolic murmurs heard loudest inside the apex, not over a large area, and not transmitted. They are seen in children of two to eight years of age, who are usually plump and well, with no cardiac symptoms. They disappear, as a rule, before the age of ten. They are important because they are often mistaken for organic murmurs on account of their musical quality. Many children are kept in bed for a long time because of the presence of such murmurs, and the parents undergo needless anxiety. Practitioners of adult medicine are most likely to make such mistakes, but these murmurs are very frequently found in children. We have called them "musical functional" murmurs for lack of a better term (see Fig. 96).

The murmurs of *congenital lesions* should not be confused with functional murmurs. Occasionally, however, although corroborative evidence, either clinical or by means of the x-ray and electrocardio-

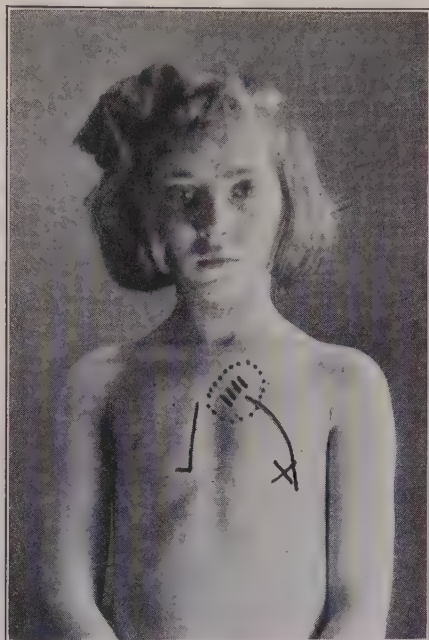


FIG. 97.—FUNCTIONAL MURMUR.

Heart

Size, action and sounds normal.

Murmurs: There is a systolic, soft, low-pitched, blowing murmur in the pulmonary area.

Thrills: None.

gram, may be lacking, the murmur is so loud that some type of congenital defect is suspected. This can only be ruled out if the murmur disappears after a period of years.

Cardiorespiratory murmurs can be differentiated by their relation to respiration. They are usually caused by the heart pushing away the air from the lung during systole, and are increased, as a rule, during deep inspiration. They disappear when the breath is held. The distinct rubbing character to the sounds caused by fibrinous pericarditis and in pleurisy should not lead to errors.

Tricuspid insufficiency can be diagnosed in the presence of the following signs: (1) A long, harsh, blowing systolic murmur heard at the right of, or over, the sternum, at the level of the fifth rib at its attachment to the sternum; (2) enlargement of the heart both to right and left; (3) enlargement and pulsation of the liver; (4) pulsation of the external jugular veins; and (5) the presence of other murmurs of valvular disease.

Tricuspid stenosis is so rare that it is seldom diagnosed. The murmur is like that of mitral stenosis but is heard to the right of the sternum.

The diagnosis of *aortic stenosis* can be made when there is: (1) A loud harsh systolic murmur at the aortic area to the right of the sternum, transmitted up; (2) systolic thrill in this area; (3) faint or absent second aortic sound; (4) a pulse with a slow rise and broad summit; and (5) a very large left ventricle and forceful apex thrust. True stenosis must be differentiated from roughening of the valve, which may give a murmur and no other signs of stenosis.

Aortic insufficiency can be diagnosed on the murmur alone, which is characteristic. It is a blowing, rather distant, faint sound and is often heard best with the patient sitting up and leaning forward. It may be loudest over the sternum at the level of the second, third or fourth interspaces, just to the left of the sternum at these levels, or in the second or third spaces just to the right of the sternum. In addition, the heart will be found to be enlarged, particularly to the left, the pulse will probably be collapsing in type, and there will be increased pulse pressure due chiefly to lowering of the diastolic pressure.

Acute infection of the *myocardium* is part of the picture of rheumatic fever, either with or without other signs of this infection. The diagnosis is based on tachycardia, changes in the form of the electrocardiogram, which indicate damage to the myocardium, and signs of

acute infection, except where the fever is clearly due to some other cause. In more severe cases of myocarditis there is evidence of congestive failure, and usually some enlargement of the heart. Since we know that the functional efficiency of the heart depends chiefly on the condition of the muscle, the necessity for making the diagnosis of acute or chronic myocarditis is clear.

Enlargement of the heart implies a combination of dilatation plus hypertrophy, since they rarely occur separately. The essential points in making the diagnosis are: (1) An apex-beat which is further to the left than normal. The normal apex is usually not more than 1 centimeter to the left of the midclavicular line. (2) Increased area of cardiac dulness determined by percussion. And (3) increased size of the heart as shown by the x-ray. In children the size and force of the apical impulse is often enough to make the diagnosis of enlargement. *Hypertrophy* of the heart gives a strong heaving impulse. In *dilatation* the impulse is apt to be diffuse, the cardiac borders are increased in area, there are murmurs indicating insufficiency of the tricuspid and mitral valves, and there may be signs of congestive failure. Dilatation is always, at least in children, associated with acute infection of the myocardium. Enlargement of the left auricle may sometimes be discovered by percussion, but is usually diagnosed by the x-ray.

The presence of a friction-sound heard over the base or the body of the heart is sufficient evidence of fibrinous, or serofibrinous, *pericarditis*. Serous effusion into the pericardial sac is shown by increase in the area of cardiac dulness, friction-sound, distant or faint heart-sounds, and the characteristic pear shape of the x-ray shadow.

In cases of endocarditis which show clubbing of the fingers and signs of sepsis, *subacute bacterial endocarditis* engrafted on rheumatic or congenitally deformed valves should be suspected. The diagnosis can be made when, in addition to signs of valvular disease, there are (1) signs of sepsis, (2) positive blood-culture (usually *Streptococcus viridans*), and (3) embolic phenomena.

Physiological.—The various arhythmias which indicate changes in the physiology of the heart action are completely covered in the chapter on electrocardiography, but a few points may be mentioned here. Sinus arhythmia is common in children and is chiefly important because it may be unrecognized. If there is any doubt whether the irregularity is of the sinus type or not, the electrocardiogram will make it clear. In this condition there is a slowing of the heart rate,

followed by a series of quicker beats. This varies with respiration, the rapid rate occurring during inspiration, the slow during expiration. Occasionally the irregularity is so pronounced that auricular fibrillation is suspected.

Premature contractions are recognized when a beat occurs before the time for the normal beat. This beat is followed by a pause which is longer than normal. Premature beats can usually be detected by palpation of the radial pulse as well as by auscultation. They are chiefly to be differentiated from "dropped beats" which are usually the result of partial heart-block. In this condition there is a pause equal to twice the length of the normal pause. In premature contractions, careful auscultation at the apex will detect the sound of the premature beat, early in the period of intermission of the pulse. In dropped beats there is complete silence throughout the pause. Dropped beats from sino-auricular standstill and from heart-block can only be differentiated by its electrocardiogram.

Complete heart-block is to be suspected when the heart rate runs regularly below 40 to the minute. Simple bradycardia has a higher rate, up to 60 per minute, and varies with change in position and with exercise, in contrast with complete block which is uninfluenced by these factors.

In *effort syndrome* there is a disturbance of function of the heart without an anatomical basis, although sometimes it is found in individuals who have organic heart disease. The signs and symptoms have already been described.

Functional.—The functional diagnosis is less easy to make than the anatomic. A good deal can be learned about the heart function by the size, and by the character of its action. The hand laid against the chest gives the examiner no small amount of information as to the efficiency of the organ. It would seem that a fairly simple pump like the heart could be tested and measured like any other machine, and consequently there has been a long search to find a simple method by which the efficiency of the heart can be rapidly ascertained. Determination of the functional capacity of the heart is important because we must be able to give directions to the parents as to the amount of activity the child can be allowed. But it is not only the heart that is involved; the whole organism is affected when the heart is damaged, and for that reason evaluation of the heart's efficiency is a complicated problem. In addition there is always the question as to the presence of active infection, either rheumatic or any other

acute disease. General weakness following recovery from any illness obviously affects the capacity of the individual to exert himself.

The anatomical and etiological diagnosis should have no influence when making the evaluation of the functional capacity of the patient. It should be the result of the analysis of the child's functional capacity at the time of the examination. Conclusions can be arrived at by careful history from the patient of the effect, as shown by dyspnea or precordial pain, of ordinary exercise such as walking or running on the level, or uphill; climbing stairs, and taking part in games. The normal activities of a child of the age of the one under examination should be compared with what the child actually does. Watching the child's reaction to exercises will aid in reaching a conclusion. In the presence of varying degrees of heart-failure, the functional capacity is usually correspondingly decreased, but at times it is surprising to see how much can be done by a child who appears to be severely handicapped.

FUNCTIONAL CLASSIFICATION

All heart disease does not fall into one category. Obviously, a clear idea of the classification of diseases of the heart is essential to any proper comprehension of the individual case. The older classification into organic and functional, congenital and acquired may serve as a broad foundation, but it is not complete; it does not go far enough. The New York Heart Association was the pioneer in the effort to make a classification which would be practical for use in the cardiac clinics. The first classification divided all cardiac patients as follows:

1. Patients having physical signs but no symptoms
2. Patients who had symptoms in the past but not at present
3. Patients with symptoms at present
 - (a) Mild
 - (b) More severe
 - (c) Heart-failure
4. Possible heart disease; physical signs but probably not organic disease
5. Potential heart disease; rheumatism, chorea, etc.

After using this for a few years, it was modified to the following:

"Class I. Patients with organic heart disease able to carry on ordinary physical activity without discomfort. Ordinary physical activ-

ity does not cause undue fatigue, palpitation, dyspnea or chest pain. Patients in this class do not show physical signs of congestive heart-failure or signs of active heart infection.

"Class 2. Patients with organic heart disease unable to carry on ordinary physical activity without discomfort.

"(a) Activity slightly limited. Ordinary physical activity causes undue fatigue, palpitation, dyspnea or chest pain. Patients in this class rarely show physical signs of congestive heart-failure or signs of active infection.

"(b) Activity greatly limited. Less than ordinary physical activity causes fatigue, palpitation, dyspnea or chest pain. Patients in this class usually show one or more physical signs of congestive heart-failure, the anginal syndrome or signs of active heart infection.

"Class 3. Patients with organic heart disease with symptoms or signs of heart-failure at rest, unable to carry on any physical activity without discomfort. There is fatigue, palpitation, dyspnea or chest pain at rest. Patients in this class show marked physical signs of congestive heart-failure or the anginal syndrome, or signs of active heart infection.

"Class 4. Possible heart disease. Patients should be so classed who show abnormal physical signs or symptoms referable to the heart, but in whom the diagnosis of heart disease is uncertain.

"Class 5. Potential heart disease. Patients without heart disease, whom it is advisable to follow because of the presence of an etiological factor which might cause heart disease, should be diagnosed as potential heart disease, Class 5. In such cases the etiological factors should be stated."

Class 1. This includes a large number of children with physical signs which make the diagnosis of heart disease certain, but who have no symptoms referable to the heart. These children have had some form of rheumatic fever with subsequent permanent heart damage, but the injury to valves and myocardium has been slight and the function of the heart remains perfect, or at least gives no subjective symptoms of inadequacy. These children usually have a mitral or aortic insufficiency with some enlargement, good heart action, no dyspnea, palpitation, chest pain or edema; and the general health and nutrition are as good as the average of children without heart disease. In this class belong the congenital cases whose lesions are slight and are often discovered by accident. An instance of this was a fourth year medical student who had a patent ductus arteriosus with a loud

murmur, and who never knew he had it until it was discovered by chance. He had played football all through college and was perfectly strong and well.

Class *2a*. This class is composed of children who have symptoms of slightly lowered cardiac reserve. They are conscious of their hearts and have symptoms of distress after ordinary exertion. They are able to go to school, walk about the streets and take part in noncompetitive games, but they cannot run far nor go upstairs rapidly without more than the normal amount of dyspnea. They may have some disturbance of heart action, too rapid or tumultuous action, or precordial pain on exertion. Cough and expectoration are not symptoms of this group, but they have small resistance to infection. Anemia and malnutrition are common.

Class *2b*. The differentiation between Classes *2a* and *2b* is an artificial one, since the degree of symptoms varies so greatly. In the milder cases only a few symptoms are recognizable—moderate dyspnea and abnormal heart rate on exertion. In the more severe group, *2b*, these symptoms are brought out by less exertion and are more marked. Judgment by different observers will classify the intermediate cases differently. There might as well be one class or several if there were accurate means of measuring the functional capacity. Many cases of Class *2b* must have some active infection; many verge very closely on Class 3. On the other hand, some are only a little worse than those of Class *2a*.

In determining the classification many exercise tests are in use, no one of which gives all the information desired. The simplest method is to have the patient do a small amount of exercise, and note the heart rate before and afterwards. Hopping on one foot, bending over and touching the floor with the hands, swinging dumb-bells, climbing stairs and deep knee-bends are the favorite forms employed. The normal heart speeds up after these exercises. The call for blood dilates the arteries to the muscles and lowers the blood-pressure, and the heart rate increases in an effort to maintain this pressure. The amount of this increase in heart rate and blood-pressure varies greatly in health as well as disease in different individuals, so that the result of a test is simply a rough indication of what the patient can do. It is usually assumed that the heart rate and blood-pressure return to normal within two minutes after any of these standard exercises, such as thirty knee-bends. More useful information can be gained by watching the amount of dyspnea produced, pallor or flushing of the face,

forceful overaction of the heart and the state of fatigue of the patient after such an exercise. If the examiner uses the same exercise consistently he learns to recognize the average reaction in a healthy individual and know when the reaction in terms of symptoms is abnormal. Such information is not exact, but is definitely a help in sizing up the functional capacity of the patient.

Class 3 includes children who have symptoms of heart-failure at rest; they have no cardiac reserve. Formerly these cases were called "decompensated," when edema and other signs of congestion were present. We recognize the fact now that the heart is failing when it is acutely infected, and this class, therefore, includes cases of acute myocarditis even without congestive failure. The variation in the severity of the symptoms depends primarily on the severity of the infection, and partly on the extent of the changes in the valves, myocardium and pericardium. Sometimes it is difficult to decide whether a child should be put in Class 3 or *2b*. He may fluctuate for months, part of the time belonging in one class, part of the time in the other. When this occurs the infection is probably always present. We have not found Barringer's delayed rise in blood-pressure a practicable test.

The functional classification is not permanent; the child may go through an attack of heart-failure and finally improve up to Class 1. This does not often occur, but it is quite common for the child to recover sufficiently to belong in Class *2a*.

Class 4. These children are mostly those with soft systolic murmurs over the pulmonic area or over the body of the heart, but have no demonstrable cardiac enlargement. Organic heart disease cannot be definitely diagnosed or ruled out. The word "possible" seems the safest term to use in these cases, since it implies that, although the physician at the time of examination does not think that true heart disease is present, he cannot absolutely prove its absence. These children must be examined periodically two or three times a year to check up on the condition, but they should be allowed to lead normal lives. After study it may be found that such a child was in reality having a very mild carditis when the murmur was first noted. The proof of this lies in the later development of cardiac enlargement or of the murmur of mitral stenosis. Some children have to be followed for a number of years before the true explanation of the physical signs can be given. It scarcely needs repeating that a systolic murmur at the apex does not necessarily mean disease of the mitral valve, even though the murmur is fairly loud and rough in quality.

Class 5 includes children who have had one or more of the rheumatic manifestations, but who, at the time of examination, have no evidence of organic heart disease. Children with a history of chorea constitute the bulk of this class, plus the few who have had rheumatic arthritis but have not yet developed heart disease. These children must be examined regularly to get the earliest sign of cardiac involvement, and also to see that they are leading wholesome, normal lives.

This classification of heart disease is useful to the physician and the social worker as a guide to treatment. It also leads to more careful and accurate diagnosis, not only of the heart lesions present, but of the condition of the child. After all, it is the child that we are treating, not the heart alone. It is entirely too easy to examine and prescribe for the heart only and entirely forget the patient. If the choice had to be made between a cardiologist and a pediatrician to care for the child with heart disease, the latter would probably produce the best results, even though he actually knew less about the cardiac condition. A pediatrician with a special knowledge of heart disease in childhood is perhaps the ideal.

CHAPTER XIV

THE PROGNOSIS

What is the probable outcome in a given case of rheumatic heart disease? This question is most important and difficult. In the opinion of some it is not worth while to educate nor to spend much money for the care of cardiac children. This was the view of one of the most famous clinicians in the early part of this century. He said that these children rarely grew up, were chronic invalids anyway, and died in early or middle adult life, so why waste time and money on them.

That point of view was all too justified in the average hospital case in those days. These children came in for a short time, were discharged as soon as the staff tired of listening to their murmurs, and went out of the hospital too soon in almost every instance. The result was an early break and a return to the hospital in a short time. This went on until death occurred at puberty or in early adult life. No one could claim that the effort and money spent were worth while. But in the last decade or more, since the study of systematic care of these children was undertaken by large numbers of organized cardiac clinics, we have come to believe that the outlook is not so hopeless. It is true that many children still go steadily downhill with recurrent attacks of polyarthritis and carditis, and even when given the best care possible in the hospital go from bad to worse. They die in a few months in spite of all we can do because the acute process goes steadily on, or recurs so frequently that the heart is progressively and hopelessly damaged.

But this is not the whole picture. Those who have worked in cardiac classes have learned that a great many children suffer only one or perhaps two attacks of rheumatic disease. The heart is only slightly damaged and seems almost, or even completely to recover from the injury. When these children are given proper care they may go on to grow and gain weight, becoming normal in size, strength and ability to do all that a normal child can do. The average child attending the Huddleston Memorial Class looks healthier and is better nourished than those in the other Bellevue outpatient classes.

The prognosis depends on several factors: The number of recurrent attacks, the amount of damage done to the heart, but most of all, on the kind of care the child gets. This does not mean medical care alone, but it includes the way in which the child lives at home, in school and at play. In other words, it depends on the coöperation of the parents with the doctor and the willingness of the child to learn how to live his life properly. The child who refuses to obey the laws of health and hygiene, who sits up late at night, eats improper and insufficient food, exercises to the point of overfatigue and in general leads an unhygienic life is an exceedingly poor cardiac risk. One sees such children who come to the cardiac class in not very bad condition, but who go downhill rapidly because they cannot be controlled. The cardiac child must, above all others, obey all the laws of health all the time and, in addition, must learn his own exercise tolerance, and live within it. The child who is docile and under good discipline, intelligent enough to understand advice and to follow it, has an infinitely better chance.

The *parent* is an important factor. Some mothers become panic-stricken and fear to discipline a child with heart disease lest the child's crying or fretting will make him worse. The resultant spoiling often merely prepares the way for a quick descent to the grave. On the other hand, most mothers when properly educated by the physician will be more careful in following directions because they realize that heart disease is a serious matter. The greater care given the child results in better health and strength and a better chance for recovery. It has been noted repeatedly in the Huddleston Class that children with heart disease who are undernourished can be brought up to normal weight more easily than other children with malnutrition who are under treatment in the Malnutrition Class or the general clinic.

The cardiac child is frequently undernourished. If his *nutrition* can be improved and if his weight approaches the normal, his heart usually improves also. The severer cases often cannot be made to improve in nutritional condition. Therefore, this ability to put on weight is *usually* a good sign, especially in the ambulant case. It is not invariable, for some of the severest cases get quite fat in bed, without much improvement in the cardiac condition.

The *type of lesion* influences the prognosis to some extent. The child with only one valve lesion has a better outlook than the one with two valves involved.

Aortic insufficiency alone has a prognosis which is rather good.

The heart becomes moderately hypertrophied and compensates for the lesion, which is often not a severe one. These children often grow to be large and strong, and up to puberty, at least, are our best specimens.

Aortic stenosis is seen only in the older patients and does not remain under observation long in a children's class, but the children have not done quite so well as the cases of aortic insufficiency. The heart is working against great resistance and one feels that the burden will probably, sooner or later, be too much.

Mitral disease varies greatly in its prognosis. Many mild cases are seen which go on for years with a definite systolic murmur, with only moderate enlargement and good cardiac function.

The cases with signs of stenosis do less well, although many of them go on indefinitely with a variable inconstant presystolic murmur and little disability.

The children with definite and constant double mitral murmurs do badly after a time, as a rule, although some live to go to work. Their cardiac function is never very good, however.

Aortic and mitral disease combined give a worse prognosis, but even here some children improve greatly and keep going until adolescence.

The advent of tricuspid insufficiency is one of the most serious things that can happen to any heart. Since it comes on late in the disease, it is usually the beginning of the end. Therefore, a poor prognosis may be given in every case with pulsating tender liver and pulsating veins of the neck.

Enlargement of the heart affects the outlook. Marked enlargement usually means a poor prognosis, especially when the apex-beat is diffuse and flopping. Enlargement in aortic disease is less serious than in mitral. The combination of a double mitral murmur with marked enlargement is a very bad one from a prognostic standpoint.

Of course, the condition of the heart muscle is the most important factor, but this we can only infer by physical examination, from the size of the heart, the character of the apex impulse and the general impression of the heart action. The electrocardiogram gives more definite information in some cases but fails us in others. When there is block or defect in the transmission mechanism the prognosis is unfavorable. Serious arrhythmias like flutter or fibrillation also give a bad outlook. But in many cases the electrocardiogram only gives us "left (or right) preponderance" which can be inferred from the

physical examination, by the size of the heart and the valve lesion; in well-developed cases.

The ability of the child to outgrow the physical signs of heart disease must not be forgotten. It is easily comprehensible that myocarditis may cease and the muscle return to normal save, perhaps, for some scar tissue. But the apparent disappearance of valve lesions is more surprising. Nevertheless, it does occur, at least as far as physical signs and symptoms are concerned. We have seen a good many children with undoubted signs of heart disease who have outgrown the condition completely so far as can be determined during life. One such boy was in a hospital at the age of four years with an attack of pericarditis and endocarditis. He was desperately sick and when removed to his home the mother was told that he would probably not live. He was seen at Bellevue Hospital in 1916, at the age of ten, with all the signs of mitral disease. His heart was moderately enlarged, the action was fair, there was a loud mitral systolic murmur transmitted well to the axilla and back. He was rather poorly nourished. No one who examined him doubted the existence of mitral disease. In the next few years he grew rapidly, his nutrition and strength improved greatly and his heart murmur gradually disappeared, although his heart remained slightly enlarged. He had no symptoms.

The late Dr. L. Emmett Holt told of a boy who in childhood had been under his care for several years with definite and repeated attacks of heart disease. As the boy grew up he developed remarkably. He went to college, played football and felt perfectly well. Dr. Holt examined him after he finished college and found no physical signs whatever.

Such cases make one feel that the prognosis is never hopeless. They enable us to hold out to the parent the hope that any child *may* outgrow his heart disease.

The child occasionally seen with hypertrophy and no valvular lesion, no hypertension, nor other cause to explain the enlargement, may possibly be accounted for by the above sequence of events. That is, he may have had an attack of carditis with valve involvement, which is later outgrown, leaving only hypertrophy.

The explanation of the disappearance of signs of valve lesion is not entirely satisfactory. The best theory seems to be that the heart grows with the child, the orifices grow and the valves grow. It is possible that this growth may so adjust itself that the stenosed valve orifice enlarges enough to become almost normal in its capacity to

allow the passage of blood, the incompetent flaps adjust themselves by growth so that they again close the orifice properly. Growth is a factor present only in the child. The damaged heart of the adult has not this hope of recovery. But the heart of a child of twelve to fourteen years is quite a different organ from the same heart at four or five years.

Even when the physical signs do not all disappear, growth is a factor not to be ignored. The murmur may remain but the heart may be a functionally perfect organ.

A man of fifty-five years was seen by one of us some years ago with an acute illness. He had a very loud mitral murmur and moderate cardiac enlargement, but lived an active, useful life. He played golf, changed his own automobile tires, ran upstairs and paid no attention to his heart. His aunt, who was present, said that he had been in bed most of the time from his fifth to ninth years with repeated attacks of rheumatic fever and acute heart disease and had been an invalid until nearly fully grown. He later had a prostatectomy with a normal recovery. Such a case gives hope for the future of even the children whose hearts seem to have been severely damaged.

A girl was in Bellevue eleven times with attacks of mitral and then aortic disease. The mitral signs gradually disappeared, she has grown up and is able to earn her own living if she takes proper care of herself. She has a definite aortic murmur, but her heart is not much enlarged and she can walk, dance a little, but has to be careful not to do too much.

The worst prognostic sign is the tendency to frequently *recurrent attacks* of rheumatic infection. The children who have one attack after another all do badly. The tonsils, teeth and sinuses may be all cleaned up. The child may have ideal hospital or home care, but if he keeps reinfecting himself, his outlook is bad. What the source of the reinfection is may not be determined. It may be merely repeated throat inflammations, or the virus or organism may lurk in the lymph-nodes, or even in the heart itself. But, whatever the source, the heart suffers repeated injury, the child gets more and more anemic, usually loses weight with each recurrence, and finally dies in an attack of congestive heart-failure.

Rheumatic nodules give a bad prognosis because they indicate a tendency to reinfection. Swift says that whenever there are rheumatic nodules under the skin there are Aschoff bodies in the myocardium, so that without evident or immediate new cardiac involvement it is certain

that there is fresh myocarditis going on in the child with a crop of new subcutaneous nodules.

Accurate data on the course and prognosis of a disease so chronic and so apt to present recurrences as rheumatic polyarthritis, chorea and rheumatic heart disease would be obtainable only if a large number of cases could be followed from the time of the onset of infection through death, *i.e.*, a period of at least thirty years. This, at best, would be an almost impossible task, and, at present, we have to be satisfied with studies over shorter periods of time. Two points are desirable to determine: (1) What are the probabilities as to recurrence, and (2) how long will an individual probably live after the primary infection. Recently, Wilson, *et al.*, made a careful study of five hundred cases of heart disease followed for periods up to ten years after their first visit to the clinic. Although definite rules for prognosis could not be deduced, the findings are suggestive of what can be expected to happen in any group of children with heart disease. Of the 413 rheumatic children in her series, Wilson states: "At the age of seven, the chances are even that a rheumatic child will undergo a recurrence the following year; of the children not likely to relapse the following year, the chances are two to three that the interval of freedom from infection will not be greater than one year. At the age of fifteen, however, the chances are only one to three that infection will recur in the following year; if no recurrence takes place at that time, the chances are about one to two that the interval of freedom from infection will be only one year." Stated in another way, "Of 172 children who have arrived at the age of seven years, and who have by this time become infected, only six, or 3.5 per cent, in our experience can hope, in the years following to remain free from infection for a period as long even as six years; only one for a period as long as ten years. Beyond this our observations do not yet reach." Twelve per cent of the children of this series died, the most common age at death being between eleven and fourteen years. Of children who had rheumatic nodules, 33.3 per cent died, death occurring in 60 per cent of these within one year. Of those living, 45 per cent have lived three or more years after nodules were first observed. The interval between the first manifestation of rheumatism and involvement of the heart was one year or less in 63.1 per cent.

The 413 children in Wilson's series experienced an average of four attacks, covering a period of two and three-quarters years, per child.

This study confirms the known fact that rheumatic fever is a

chronic disease with a marked tendency to recurrence. Individual attacks vary from a few weeks to several months. Many cases showing this could be cited from our experience at Bellevue Hospital. One child, aged five years, admitted in June with a history of several weeks of joint pains, fever and dyspnea, was still on the ward in the following March. During this time she experienced several attacks of pericarditis, many crops of subcutaneous nodules, showed congestive failure of the heart three times, and was constantly dyspneic and orthopneic in varying degrees. At no time while in the hospital did she have a polyarthritis. She was fever-free only a few days. This child represents the very severe type of infection which manifests itself mainly as a carditis, and which obviously has a very grave prognosis. In contrast to this child is one who, at the age of three, was admitted to the hospital with well-developed heart disease, joint pains and congestive failure. After several months of hospitalization, and an up-and-down course, she was removed from the hospital against advice. Her death was assumed, particularly as the family moved from the address given, until she appeared in the clinic two years later. She had varied from periods of moderate physical activity to periods of being bed-ridden, and was kept on small doses of digitalis for months at a time. At present, she is in surprisingly good condition. In other words, it is impossible to state exactly in regard to any child what the probabilities are. Beyond puberty the chances of rheumatic infection appear to decrease, and it has been suggested that immunity develops at this time of life.

In regard to the question as to life expectancy in the individual, an absolute answer is impossible. Wyckoff and Lingg, in their analysis of about one thousand cases of heart disease, show that there is a marked drop in the number of rheumatic cases after the age of forty. The obvious explanation for this is that deaths from rheumatic heart disease occur chiefly before the age of forty years. The length of time intervening between time of infection and onset of heart-failure has a bearing on prognosis, and the time between the onset of rheumatic fever and the development of definite organic heart disease is also important to know. Unfortunately, few accurate data are available bearing on these points. Wilson, *et al.*, found that 63 per cent of these children showed marked cardiac involvement within one year of the onset of infection. It is fair to state that heart disease will probably develop in one to six years after the initial infection, but that exceptionally many years, even life, will pass without any heart involvement. Heart disease

in this connection refers to permanent anatomical changes in the myocardium or valves or pericardium.

Romberg states that heart-failure begins, on an average, seven years after the development of heart disease. The stage of failure, he says, may last on the average four and eight-tenths years. Cohn deduces from analysis of various figures that "the disease will have run its course between the ages of thirty-five and forty-five."

The table from Coombs shows the results of his study:

- 5.1 per cent died in the first year after onset.
- 11.2 per cent died within five years after onset.
- 21.4 per cent died within ten years after onset.

TABLE VIII
AGE INCIDENCE OF DEATHS (COOMBS)

Ages, Years	In Clinical Series, Per Cent	In Autopsy Series, Per Cent
Up to 10.....	3.6	8.1
11 to 20	30.9	30.6
21 to 30	24.2	24.4
31 to 40	21.8	14.2
41 to 50	13.3	13.2
51 upwards	6.0	9.1
Average age at death	28.6 years	28.2 years

In general it can be stated that rheumatic fever is a chronic disease which attacks chiefly children of school age, that the heart is involved in a high percentage of cases, that there is a marked tendency to recurrence, and that life expectancy, which is modified by the severity and number of attacks and the age at which the first attack occurs, does not extend much beyond forty years of age.

CHAPTER XV

TREATMENT OF HEART DISEASE

The first step in the treatment of a patient with heart disease is to ascertain that the diagnosis is accurate. The medical profession has made far too many mistakes in omitting this essential. Children with functional murmurs or mild congenital lesions are kept in bed for months or years when the heart is really normal from the functional standpoint. Other children who have had insignificant joint symptoms or a very mild chorea are allowed to run and play, or are sent to school when there is an early endocarditis or an active rheumatic myocarditis. It is therefore necessary to be perfectly sure as to the real condition of the heart, in order not to work an injustice to the individual. Not every murmur means that rest in bed is needed. Simple irregularities (sinus arrhythmias or premature systoles) do not indicate heart-failure. But a real mitral murmur developing after a rheumatic polyarthritis or chorea should be recognized, and the heart guarded and watched carefully for a long time.

The treatment of heart disease must be considered from several standpoints. Let us assume that the diagnosis has been complete and accurate. Our treatment is guided in the first place by the functional capacity of the heart; by its exercise tolerance or cardiac reserve. It is obvious that a heart with no reserve force, no exercise tolerance, needs rest. This can be given only by rest in bed, and this must be continued as long as the function of the heart remains much impaired. There are different degrees of this condition. A patient may have no exercise tolerance, yet be relatively comfortable in bed, while another with complete heart-failure is dyspneic to the point of orthopnea, restless, suffering even after confinement to bed for months. It is safe to say that bed care is demanded for all patients in whom slight exercise, walking on the level, causes disturbed heart action and dyspnea. This bed care should be continued until the heart muscle has recovered part of its strength and has acquired some reserve.

The first stage of the disease begins with the first attack of rheumatic fever, whether in the form of chorea, polyarthritis, growing

pains or carditis. All cases should be treated as though the heart were involved, that is, bed care should be continued for at least two to three weeks after the acute phase has apparently cleared up. The leukocyte count and the temperature should be normal. Sometimes, slight rises in temperature or a persistent leukocytosis with an absence of fever may be traced to infected tonsils, teeth or sinuses. If these foci are eliminated the temperature and leukocyte count in some cases return to normal. When all other reasons for fever have been ruled out, the evidences of infection must be considered as due to active inflammation of the heart. By fever we mean a rectal temperature which is over 99.8° F. Mouth temperatures are not reliable. In considering the significance of a temperature, the factors, such as recent ingestion of a meal, or unusual physical exertion, which may normally cause a slight elevation in the temperature, must be considered. If there is doubt, the child should be kept in bed at rest for a day or two and the temperature taken at two-hour intervals to detect any significant rise.

The question of the treatment of heart disease should be considered from several points of view: First, from the point of the infections present; second, direct treatment of the heart by drugs and other means because of the disturbed physiology due to mechanical difficulties; third, prevention; and above all, first and last, the care and handling of the child.

Infections have already been considered in the descriptions of the various forms of rheumatic fever. The importance of eliminating all foci of infection cannot be emphasized too strongly.

The following cases are instructive:

A colored boy, ten years of age, was admitted to Bellevue Hospital with an old rheumatic heart and congestive failure. He was not in great distress, but the usual measures did not control the edema and other physical signs of failure. After several weeks he developed swelling of the lymph-nodes at the angle of the jaw and it was found that a tooth was infected. Following removal of the tooth, and hence elimination of that focus, there was prompt and dramatic cessation of the symptoms.

Another child, a girl of eight years, was admitted for slight fever, organic heart disease and rapid pulse with consequent discomfort. Under rest in bed she improved subjectively, but slight rises in temperature, up to 100° to 101° F., persisted with a constant moderate elevation of the leukocyte count. Her maxillary antra were found to

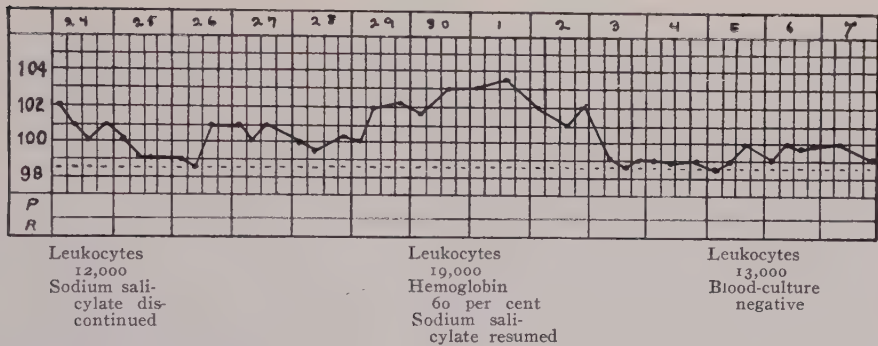
be infected and large amounts of pus were washed out. Rapid improvement in the child's general condition followed treatment of the sinuses. The temperature remained normal, the number of white blood-cells dropped and very soon she was discharged from the hospital. She had had very slight nasal discharge and no symptoms pointing to infection in this locality. Such cases show the importance of careful and systematic search for any possible focus of infection, even when there are no symptoms pointing to any particular area.

The essential part of treatment of a case of mild carditis, with slight fever, leukocytosis and rapid heart rate, is rest in bed, together with measures to improve the nutrition and the eradication of any infected focus which can be reached. Theoretically, the complete rest gives the heart an opportunity to recover from the damage done. Recently, it has been suggested by Levy that the x-ray therapy over the infected heart may help to reduce the reactivity of the cardiac tissue. The value of this procedure has not yet been sufficiently proved to justify advocating this measure, but further investigation along this line may be worth while. Many other measures are being tried out, such as induction of high fever through the use of foreign protein injections, but so far the results are not conclusive.

Salicylates, as has already been pointed out, are of great value in controlling the symptoms and pyrexia of rheumatic fever. A case in Bellevue demonstrates very nicely the effect of salicylates on the symptoms of rheumatic fever complicating heart disease. This girl was admitted with high irregular fever, dyspnea, and precordial pain. She was an extremely ill child. She had an acute fibrinous pericarditis, pulmonary signs probably due to rheumatic pneumonia, and an enlarged heart with mitral insufficiency. She had joint and muscle pains at various times. Cough was a prominent symptom, although she had no signs of congestive failure (Chart V). She was put on large dosage of sodium salicylate with improvement in symptoms and lowering of the temperature. After a time it was thought wise to discontinue the drug; within forty-eight hours the temperature began to rise again and the child became more uncomfortable. Salicylates were resumed with prompt lowering of the temperature. Later, the same thing was tried again with the same results. Joint pains were not a marked feature of the case, so that the effect of the drug on the other symptoms was striking. Although we do not feel that salicylic acid has a specific bactericidal action in rheumatic fever, its value if used in large doses in such cases, cannot be overemphasized. The drug can

be withdrawn gradually by reducing the dose each day. If there is no recurrence of symptoms after a few days without medication, it can be omitted permanently. It is wise not to allow the child any physical activity until he has been without the drug and without a return of symptoms for several days. When the child first gets out of bed, the effect on the heart rate should be noted carefully. If the rise is too great and if it does not lower promptly after return to bed, he should probably be kept quiet for a longer time. Increase in exertion should be very gradual to avoid recurrence of the disease. The exact time when a child should be allowed up is almost always difficult to decide,

CHART V
EFFECT OF SALICYLATES ON TEMPERATURE



R. K., female, aged 9 years

Diagnosis

1. Rheumatic carditis
2. Mitral insufficiency and stenosis
3. Pericarditis
4. Rheumatic pneumonitis

except in cases of arthritis of the monophasic type without demonstrable heart involvement. Sometimes the past history of the case will aid in coming to a decision. If the child has previously gone through severe attacks of rheumatic fever, or has had recurrences within a short time of former attacks, great caution is indicated in the resumption of physical exertion.

The value of convalescent care is emphasized in another chapter, but it can be said here that when the home environment is poor or in need of adjustment, the child should be sent away to the country for a time. If the heart has not been permanently damaged, a few weeks or months spent in any convalescent place, not necessarily a cardiac home, is advisable. Some convalescent homes have reported good results on the general condition of the children from the use of ultra-

violet light, or from direct sunlight. All means to improve the nutrition of the children should be used.

The heart, the endocardium and myocardium of which are acutely infected, is less efficient in its action than the normal heart, and can, therefore, be called a failing heart. The danger of dilatation of the walls of a heart in such a state is always imminent and a heart once dilated probably never returns entirely to its previous condition. Congestive failure in children is always associated with active infection of the heart, and this fact affects the prognosis very greatly. The first few attacks of congestive failure can usually be controlled by bed rest, but if the infection is a very severe one, the body will be unable to combat it successfully. Control of the signs of failure, however, doubtless relieves the heart of a burden and thus assists in recovery from the attack of infection.

Treatment of heart-failure depends somewhat on the degree of failure present, but absolute rest is always indicated as the first step. Codein in adequate doses ($\frac{1}{8}$ to $\frac{1}{2}$ grain repeated every four hours) is usually sufficient to quiet the child's restlessness and to relieve the pain and discomfort and thus rest the heart. If codein is ineffective when given in repeated reasonable doses, morphin is certainly justified. A child with an acutely decompensated heart is one of the most pathetic pictures to be seen on a children's ward, and gives the physician the feeling that any means to relieve the distress, even the administration of habit-forming drugs, is only humane. When the child is going through one of his first attacks of failure, he has more chance of pulling out of it, and consequently more caution should be used in giving more than small amounts of morphin and only a few doses. An older child who has had previous attacks of congestive failure and who, therefore, is less likely to recover from the present attack, can be given morphin more freely with the idea of making his last days more comfortable. After seeing a good many of these cases, it becomes possible to tell fairly accurately the chances of recovery for the individual case.

When there is an acute fibrinous pericarditis present with pain, an ice-bag over the precordium gives a little relief to some children. The ice-bag should be applied in a jacket containing a pocket in order to hold it over the heart. Younger children are often more bothered than helped by it. In others, counterirritation in the form of mustard plasters will help, but usually this type of treatment is ineffective in relieving the pain, and drugs must be used.

Cardiac stimulants are indicated in heart-failure and digitalis is by far the most useful.

The indications for digitalization are: (1) Congestive failure, *i.e.*, edema, ascites, enlargement of the liver and pulmonary congestion. In these cases the prompt disappearance of edema and ascites, and the large output of urine are often quite dramatic. The child usually feels better, and the cough is relieved, sometimes within twelve hours, before other evidences of improvement ensue. (2) Auricular fibrillation always calls for digitalis, and the disappearance of the pulse deficit is proof enough of the efficiency of the medication. (3) Milder cardiac symptoms, cough, some dyspnea and a rapid overacting beat, but without auricular fibrillation or acute congestive failure. The signs of improvement in those in the third group under digitalis therapy are less definite, and more difficult to define than in those in the first two groups. In this group the pulse is frequently reduced, dyspnea diminished and the child's whole condition improved by digitalization. The color may become better, and he is subjectively almost always more comfortable. Ambulatory cases classed as *2b* are often strikingly helped with small doses of digitalis. Many lives are certainly prolonged and others saved by the proper use of digitalis, but the time frequently comes when the heart is so severely damaged that even digitalis cannot help. Cases which do not respond to adequate digitalis therapy have a bad prognosis.

The preparation of digitalis used is of prime importance. A reliable stable tincture is rare, and results obtained with the tincture are uneven. Most drug-store tinctures are worthless. The drug used at Bellevue Hospital on the children's service is the powdered whole leaf in tablets, supplied by the Heart Committee of the New York Tuberculosis and Health Association. These tablets are made in doses of 0.05 gram ($\frac{3}{4}$ grain), 0.1 gram ($1\frac{1}{2}$ grains), and 0.2 gram (3 grains). The cat-unit strength is known (0.1 gram equals one cat-unit) and the digitalis is known to be active and uniform in action. For rapid digitalization in an attack of severe failure (with or without signs of congestion) the following method (Eggleston's) is used: The weight of the child in pounds is multiplied by 0.015 to obtain the total amount in grams, necessary to produce digitalization. Half of this dose is given immediately by mouth; six hours later one-quarter of the dose; six hours after the second dose, one-eighth of the calculated amount, and the fourth dose of one-eighth is given after another six hours if no signs of digitalis intoxication have occurred.

For instance, if the child weighs 40 pounds, his full dose would be 0.6 gram. Approximately half of this dose, 0.3 gram, would be given as the first dose, 0.15 gram as the second, and 0.075 gram as the third and fourth doses. The total given in twenty-four hours would thus be 0.6 gram, which is the calculated full dose. If no signs of digitalis intoxication are present, the child is put on a daily maintenance dose, usually 0.05 gram three times a day, with at least six hours between doses, or 0.1 gram two or three times daily. We have been unable to formulate absolute rules for the amount of the maintenance dose, as the rate of elimination apparently varies considerably with the individual. Results with this method and of using this kind of preparation of digitalis have been uniformly good, and make us feel strongly that the statements made by others that children require from 50 to 100 per cent more digitalis than adults, must have been based on observations made when some unreliable or inactive preparation of digitalis had been used. Overdosage of digitalis has its dangers, but when properly used, it is the most useful drug we have to treat heart-failure. A patient should never be digitalized by the rapid method if he has been taking digitalis recently.

While the child is getting these large doses of digitalis, the pulse should be watched closely and counted frequently by the intern or by a carefully instructed nurse. In none of our cases has the heart rate fallen below 70, although complete heart-block may occur as the result of digitalis. Early signs of digitalis intoxication are premature contractions and vomiting. If either of these occurs, digitalis should be stopped temporarily. Vomiting may be the result of heart-failure, and is controlled by digitalization, but if it recurs, after the whole, or most, of the calculated dose has been given, it is probably caused by the digitalis. The drug can be omitted for forty-eight hours, and the maintenance dose then begun. If the child is vomiting before digitalization so that he is unable to retain anything by mouth, the tablets may be mixed with water and given by rectum, or one of the reliable intravenous preparations may be used. Frequent electrocardiograms should be taken when a child is given digitalis over a long period.

The results obtained with this form of digitalis and this method of administration are so prompt that a failure of response points to failure of the child to take the pills. One little girl who had always reacted well to digitalis showed no effect even after much more than her required dose had been ordered for her. On investigation, a pile of the tablets was found tucked away in her bedside stand. How she

had managed the sleight-of-hand when the nurse was standing over her, was a mystery.

The ordinary drug-store tincture is practically worthless because of its unreliability. When, in addition, it is given in very small doses, there is small wonder that no results are obtained.

Digipuratium or digitan tablets have, in our experience, been the most satisfactory substitutes for the Heart Committee tablets. The ampules for intravenous use also seem to be reliable and potent in extreme emergency.

We have used various other preparations but with little satisfaction.

We have not used spartein, strophanthin, or scillaren, often enough to judge of their value. Digitalis seems to be sufficiently reliable to serve its purpose. There is an advantage in sticking to one drug and becoming thoroughly familiar with its use.

Occasionally, there are patients who still retain some of the ascites or edema, even after complete digitalization. Such cases are difficult to treat, and occur only in very severe failure. Potassium citrate, up to 10 grams daily, may help, and a salt-free diet seems to be of some aid. Diuretics which act directly on the kidney, such as caffein, theocin and diuretin, are useful in some cases. A recital of the treatment of an extremely sick colored boy, eleven years old, will show the measures which can be taken to give relief, and also the futility of the procedure as judged by the outcome of the case when the infection is overwhelming.

On admission, this child had tremendous ascites, edema of the extremities, and both pericardial and pleural effusions. Rapid digitalization with a urinary output of 2500 c.c. in twenty-four hours, removed the edema and gave great subjective relief. Some of the ascites remained. He had premature contractions after a time so that digitalis had to be discontinued. Edema began to reaccumulate, so theocin, 3 grains for three doses at two-hour intervals, was given, with an output of 3000 c.c. of urine. Maintenance doses of digitalis were started, but the edema and ascites (which had never completely disappeared) returned. He was put on a salt-free diet, and potassium citrate was given. A few days later theocin was given in the same way as before, but with no result. He became so uncomfortable from the ascites that his abdomen was tapped and 5000 c.c. obtained. At about the same time the amount of fluid in the pericardium seemed to be embarrassing the heart action, wherefore the pericardium was

tapped on two occasions, and from 100 to 200 c.c. obtained each time. For a while these measures gave the boy such relief that the outlook seemed a little brighter. In the end it was the rheumatic infection which struck the final blow, as shown by the development of an acute fibrinous pericarditis with a loud friction-sound heard very extensively. He also developed a mural thrombosis in the left brachial vein. He died with few signs of congestive failure. The measures used for this boy certainly made him more comfortable, and most assuredly prolonged his life by several weeks, but they were inadequate in the face of the overwhelming infection.

This child has been one of the few in our wards from whom removal of the pericardial effusion has been indicated. Usually the amount of fluid is not sufficient to embarrass the heart action; the interference comes later when many adhesions have formed. The place of choice in tapping the pericardial sac is posteriorly on the left side, a little below and to the left of the angle of the scapula. It has been shown that, when there is much fluid in the pericardial sac, the lung posteriorly is pushed away, so that at this point the needle will go directly into the pericardial sac without piercing lung tissue. Occasionally it is better to go along the left side of the sternum, but this is more dangerous because of the possibility of going through a blood-vessel.

Tapping of the abdomen is seldom necessary, but is indicated when there is so much ascites that the child is uncomfortable from it. Medication should be thoroughly tried first, as a child is usually very apprehensive about any procedure such as this and may be so emotionally upset by it that he collapses after the procedure is over. The technic is the same as that used in an adult.

Pleural effusion in rheumatic fever, particularly in the presence of pericarditis, is not uncommon. It occurs usually on the left side, but may occur on the right. Little discomfort is caused by a moderate amount of fluid, but larger amounts which cause cough and dyspnea may be removed easily and with great relief to the patient. Small amounts are readily absorbed.

In congestive failure, also, fluid may collect in the pleural cavity as a result of the venous congestion, rather than from inflammation of the pleura and lungs. If the pleural fluid does not disappear under drug therapy and rest in bed, it may be removed.

As we have said in another chapter, the point of view in treating heart disease in children has changed markedly in the last ten years.

The fact that the disease is a result of infection; that the infection may be persistent with few signs over a long period of time makes a tremendous difference in handling cases. Instead of saying, "Is the heart compensated?" we say, "Is the infection still active?" Instead of saying to the child, "Do not do this and that; you have heart disease," we say, "You have heart trouble, but even so, you can do this and that, and we want you to be just as active as is good for you." The emphasis is put on encouragement, rather than discouragement. This policy not only helps keep up the morale of the child and the parents, but also, we feel, gives better results in the child's condition. His physical capacity should be determined as accurately as possible, and he should be urged to live up to his capacity. This may be done by carefully graded and supervised activities. Children in Class 1 can lead a perfectly normal life. Those in Class 2a can exercise and play quite freely, but should have an extra daily rest and should probably not engage in very energetic competitive games. Children in Class 2b are greatly restricted in their activities and usually do not have to be urged to lead a quiet life; they are limited by their own symptoms. They verge closely on Class 3, and many of them must have active infection present, even though it cannot be proved.

One of the questions which had to be decided most often in the handling of children with heart disease, is how much physical activity shall be allowed. As soon as the diagnosis is made the parent immediately wants to know what the child may do. The answer depends not only on the condition of the heart but also on that of the child. Cases which belong in Class 3 can take no exercise because they have symptoms of failure while at rest. Those in Class 2b who have dyspnea, fatigue and other symptoms on less than ordinary exertion must be most carefully judged. No universal rule can be laid down, since no two children are alike. There are many who can do very little, perhaps walk about the room a few times a day or merely to the toilet. These are usually convalescent from recent rheumatic infection, with hearts considerably damaged, one or more valve lesions, definite enlargement, and heart action of only fair quality.

As the recent attack fades into the background, the child can do more and more and may be transferred into Class 2a. The symptoms of this class are chiefly more than normal dyspnea and disturbed heart action on ordinary exercise. These are difficult cases to regulate. They need a certain amount of exercise to tone up the heart muscle, for it needs to be used to its normal capacity just as much as a normal

heart, lest it become soft and flabby like any unused muscle. This group of children have hearts which are somewhat enlarged, with fair force and rate, and lesions of varying degree. Each individual must be studied with the greatest care. The natural tendency of most children to run and play to the limit of the exercise capacity insures that they will take enough exercise. The danger often is that they will take too much. This does not mean too rigid restriction. Such children can walk on the level if they do not hurry; they can climb stairs slowly, by resting after every flight. They should not run nor hurry upstairs. The older view forbade all exercise to cardiacs, lest the heart be strained. The new point of view attributes symptoms of heart-failure to fresh infection and discards physical activity as the cause of symptoms. The truth probably lies between these two extremes. Attacks of congestive failure in children are probably always due to fresh infection, but if a child has symptoms on exertion it is Nature's warning that the exercise tolerance is lowered. No one would advise a normal person to go ahead and exercise up to the point of abnormal dyspnea and rapid, tumultuous heart action which does not return to normal for some time. Therefore, a cardiac should not be allowed to take that smaller amount of exercise which exceeds his tolerance.

The normal person out of training can easily strain his general muscular system by overexercise, resulting in extreme fatigue and soreness on the next day. His heart muscle can be strained, with more difficulty, it is true, causing acute dilatation. The individual with heart disease can do the same thing but much more easily. Therefore, children with symptoms on exertion should be warned not to run, and especially not to go upstairs too fast. The exact amount of walking must be prescribed in the more severe cases, while the milder ones may walk all that is necessary within reason.

The child must not be forgotten in thinking of the heart. The amount of exercise allowed depends on the whole muscular development and nutritional state almost as much as on the symptoms caused by exercise. Thin, anemic children should never be allowed to exercise too much and should have extra rest periods during the day. Breakfast and supper in bed help to shorten the day and a good rest of one to two hours before or after the midday meal reduces the hours of activity.

Unfortunately, the lack of a reliable, easily applied, functional test makes it impossible to be scientifically accurate in prescribing exercise. The mother should be told to watch the child carefully to

see if he becomes unduly short of breath after each bit of activity. It is perhaps safe to say that there should be no objective dyspnea, nor subjective shortness of breath for more than a few minutes following exercise. The mother may be trained how to feel the heart action and also how to count the pulse. It is a good rule to decrease activity in a child who is not gaining weight, especially if he is markedly undernourished.

A child who gains weight regularly, who looks well and who has no abnormal dyspnea nor disturbed heart action after exercise may be allowed to do practically anything a normal child can do. This describes the children in Class 1.

It is not always easy to be sure about the matter of symptoms. In addition to the difficulty inherent in all subjective symptoms we have the anxiety factor injected into the mother's observations due to her dread of heart disease. Furthermore, dyspnea is a physiological symptom in all individuals after exercise and the same is true of rapid and overforcible heart action. Every child who runs on the level or up several flights of stairs gets out of breath and becomes more or less conscious of his heart action. It is almost too much to expect a child or his mother to judge accurately between the normal and the abnormal in this. How, then, can we judge of exercise for ambulant cases? The physician with judgment soon learns by experience and observation about how much a given child can do. By careful physical examination no small amount of information about a heart is gained. The size, character of the apex thrust, the extent of damage to the valves, the reaction to simple exercise tests, and determination of the vital capacity give a fair idea as to the exercise tolerance of the child.

Systematic graduated exercises for cardiac children have been tried in several classes. The idea was based on the mild, graded exercises given adults at Nauheim, Germany, and elsewhere for chronic myocardial weakness. The plan has not been particularly successful with children. The natural tendency of children to get exercise from their instinctive play insures that they will get plenty of exercise as long as they are out of bed. Many get exercise in bed if not constantly restrained. It is amusing to see a group of children who have just been put through carefully graded mild exercises in a group, do much more violent things as soon as the class is over. The only field for these very mild movements of arms and legs would seem to be in children just emerging from Class 3 to 2*b* or 2*a*. This phase of treatment needs more study.

Although in some cases, like the one cited above, the labor seems to have been in vain, yet in the long run the improvement in the way of treating these children, both ward and ambulatory cases, must be effective in saving and prolonging lives.

It is very easy to talk glibly of the *prevention* of heart disease, but actually we are working rather in the dark. For many reasons already discussed, rheumatic fever is treated as an infection which is induced or fostered by environmental factors. Emphasis is, therefore, placed on effort to change and control these factors, and therein, at present, lies our hope of the prevention of heart disease. In children, certainly, the problem is the prevention of rheumatic fever; if this can be prevented or controlled, the incidence of heart disease in children will diminish. Organized and systematic care along these lines is the logical course to pursue and presents greater hope of success than care of the already greatly damaged heart.

For several years tonsillectomy has been done routinely in some clinics with the idea of preventing the recurrence of attacks of rheumatic fever, and good results have been claimed. Unfortunately, few dependable studies have been made of this question. Wilson, *et al.*, recently examined the records of 413 children. In 247 the tonsils had been removed. They were unable to demonstrate any marked effect on recurrence of rheumatic infections after the operation. They point out that at different ages the expectation as to recurrence varies. In their series children below nine or ten years of age had frequent recurrent attacks whether or not tonsillectomy had been done. On the other hand, in older children, recurrent attacks became less frequent regardless of enucleation of tonsils. This is the only series in which the factor of age at operation was taken into account and seems to show that it should be considered in any study bearing on the question of the value of tonsillectomy.

Although from the point of view of the effect of recurrence the value of tonsillectomy has perhaps been overrated, infected tonsils should be removed from the rheumatic as well as the non-rheumatic child. The signs of infection of the tonsils are: (1) Injection of the anterior pillars of the fauces; (2) persistent enlargement of the anterior cervical lymph-nodes which drain the tonsils; and (3) follicular exudate which can be easily seen or which can be expressed from the tonsils. The embedded tonsils, which have been frequently inflamed and which appear small, are often much more of a menace than the very large tonsils. But large irregular tonsils and especially those

subject to recurrent tonsillitis should probably always be removed. Obstruction to nasal breathing is, of course, a very good indication for operation.

If the present conception of rheumatic fever as described in Chapter II is correct, then the importance of eliminating the tonsils as a possible source of the substance which is changing the reactivity of the body tissues is obvious. When the infection is not confined to the tonsils, but includes the sinuses as well, the job is only partly done when the tonsils are removed. Complete enucleation may be accomplished, but the patches of lymphoid tissue which remain in the pharynx may grow and become infected, or there may be a regrowth of lymphoid tissue in the tonsillar fossa. There can be no doubt that this regrowth occurs nor is there any reason why it should not do so. The tonsil is merely a lymphoid infiltration of the submucosa, and it is perfectly possible for new patches of lymphoid tissue to be laid down at any time in any submucosa. We have seen undoubted instances of regrowth repeatedly in children who have had perfect operations. The throat specialist who says that this is impossible if the whole capsule is removed has not made the opportunity to follow his cases over a period of years and moreover does not understand the histology of the tonsil. The capsule is nothing but a condensation of the submucosa, not different from the part left behind.

Tags of tonsil, especially in the lingual angle, are very likely to enlarge rapidly after operation, and these frequently become infected. A recent study has shown that, in a series of adults, cultures from these tonsillar remnants contained greater numbers of pathogenic bacteria than are found in the original tonsil. Such tissue may be a dangerous focus to the individual and in certain cases removal is advisable.

The time to perform the operation is sometimes a debatable question. The procedure at Bellevue Hospital in the Children's Medical Division is to wait until at least two weeks after the acute symptoms of rheumatic fever have subsided, and the child is in good condition to stand the operation. Occasionally the temperature does not become normal until after the tonsils have been removed. Each case must be decided on its merits and there are times when *noli me tangere* seems the wisest course. After the operation the child should be kept in bed for *at least one week* or longer if there is cardiac involvement. Cases of rheumatic polyarthritis and chorea sometimes appear to be precipitated by tonsillectomy, but, nevertheless, the reasonable plan is to

remove the diseased tonsils as soon as the patient can stand the procedure.

No operation should be done during an attack of acute chorea, since the child may be made much worse and even go into a maniacal state ending fatally.

The improvement in the general condition following tonsillectomy is sometimes very striking. The appetite improves, the child gains weight rapidly, his color gets better and the number of colds and sore-throats diminishes. The health of so many children is benefited greatly enough to justify the procedure in most cases, although it is impossible to say to what extent any particular child will be helped, so far as the recurrence of rheumatic fever is concerned. Our impression agrees with that of St. Lawrence that recurrences are probably less frequent if all cases are considered. In the severe cases with an established tendency to relapse one can expect less.

Teeth should be cared for to prevent development of infection of gums or teeth. Nasal sinuses should always be eliminated as the possible source of infection.

Research along bacteriological lines and development of vaccines or sera with which to treat rheumatic fever also are holding out promise of success, but at present the prospect continues to be a long, arduous uphill fight. Only if we keep in mind the ultimate objective—the saving of the lives of thousands of children—and all that has been accomplished in controlling other diseases, will we have the courage and the patience to continue the fight.

Finally, it must be repeated that the care of the child is more important than the care of the heart. A carefully planned daily regimen and diet should be given every parent at the beginning of the treatment. Directions should always be written out in full and explained most explicitly. But this is not enough in ambulant cases. At each return visit inquiry should be made to determine how well directions are being followed. It is perfectly futile to expect improvement in any child who is sitting up too late at night, who is eating an improper or insufficient diet, whose bowels are irregular, who has too little air, sun, exercise and bathing. Constantly reiterated health habit instruction is after all the physician's most potent and important means of treatment.

CHAPTER XVI

SYSTEMATIC CARE OF CHILDREN WITH CARDIAC DISEASE

Any disease which constitutes a definite menace to the public warrants well-organized, intelligent study and care. The war against tuberculosis, a disease for many years a leading cause of death, apparently aided in the general reduction of both morbidity and mortality rates. To what extent the work done in fighting tuberculosis affected these rates is impossible to state, since the decline in tuberculosis had been going on for a number of years before organized attention was given to the problem, but at least the death rate fell more rapidly after the organized campaign had begun.

Organization against rheumatic fever is made difficult because it is not a reportable disease; figures given for the increase of heart disease among the population as a whole are estimations based on reports of deaths and on hospital statistics. Accurate knowledge of the extent and distribution of the disease is not available, and will not be available until it is made reportable, just as are tuberculosis and scarlet fever and other communicable diseases.

Certain communities, particularly cities, are known to have large numbers of patients with heart disease. Such communities should make every effort to control the spread of rheumatic fever and treat effectively those individuals who already have developed heart disease. Since rheumatic heart disease affects chiefly the poorer section of the population, the question will be considered from the point of view of the clinic.

Education of the lay public as well as of physicians is the foundation of successful public health work, and it is to organizations such as the American Heart Association, local heart associations, and city and state departments of health, that we can look for propaganda designed to spread information.

Systematic care of cardiac children, and particularly the "potential" group, is an essential part in the scheme to reduce the incidence of heart disease.

CARDIAC CLINICS

As in tuberculosis the class or group method has been found to be invaluable for the care of children with heart disease and for the follow-up of rheumatic fever and chorea. Formerly these children drifted through the outpatient departments of hospitals with desultory care. There was no mechanism to handle them in a systematic way. Guile's class for adults was founded in 1912. In 1915 and 1916 the first children's cardiac classes were organized at St. Luke's Hospital by St. Lawrence, and at Bellevue Hospital by Smith. They were started in an effort to develop a system by which these children could be watched and treated along the lines of tuberculosis classes.

The ideal cardiac clinic is connected with a hospital and is manned by a medical personnel with knowledge of and interest in heart disease. The social service staff of a cardiac clinic should have adequate understanding of the fundamental problems.

Source of Patients.—The case load of a clinic is made up of children from many sources. Some are referred from the general clinic when heart disease has been discovered. Many are discharged from the wards of the hospital, some are discovered in the yearly examinations given in public schools. Others may be referred by social workers who suspect heart disease, or who have happened to learn that the child had rheumatic fever. These should go through the general clinic first because only a fraction will really be found to have heart disease. The heart clinic is a special clinic and should carry on its case load only individuals who have definite heart disease, who have had some form of rheumatic fever, or in whom there is a real diagnostic problem. It should not carry the burden of initial examination.

The case load should be checked up to make sure that only active cases are carried on the roster. This means that every case should be carefully studied and completely analyzed when first admitted to the clinic, or else there will be carried on the records cases which do not rightfully belong in the clinic. At least one cardiac clinic (the adult cardiac clinic at Bellevue Hospital) does not register a patient and include him in the case load until three visits have been made. All special examinations such as Wassermann, x-ray, electrocardiogram, etc., have been done, and the data are sufficiently complete to make a satisfactory diagnosis, etiological, anatomical, and functional, before the case is admitted to the clinic. One advantage in this plan is that a patient who attends a clinic three times has indicated that he really

wants to be cared for in that clinic and that he is not merely a "shopper."

Organization and Administration.—Certain minimum standards for an ideal cardiac clinic have been drawn up by the Heart Committee of the New York Tuberculosis and Health Association. These standards are a guide for anyone who wishes to establish a clinic for the care of individuals suffering from heart disease. Each clinic should have a chief-of-clinic who has, in addition to wide clinical experience, special knowledge of the problem of heart disease. He should be responsible for the quality of the medical work done, and for the organization of the clinic. He should see all new cases to confirm the diagnosis and should be consulted by other members of the clinic staff when there is any problem of diagnosis or therapeutics. An arbitrary standard, based on experience, of one physician for every fifty patients in the clinic, is a practical and desirable basis for establishing the medical personnel. It is obviously necessary that the physicians should attend regularly and put in sufficient time, otherwise the patients will not receive adequate attention. The only way to do this is to keep an accurate record of attendance, the actual time spent by each physician, and the number of patients he sees during each period.

Since in most clinics patients who need hospital care are referred to the wards of the hospital with which the hospital is connected, it is wise to give ward appointments to at least part of the clinic staff. All members of the staff should have ward privileges, since it is only by following cases through every phase of their progress that a real conception of rheumatic fever can be obtained. Interns should be assigned to spend a certain amount of their time in the cardiac clinic as part of their training. They will then learn why cases are referred to the hospital, which they would otherwise feel were "cluttering up the ward." This attitude is particularly apt to be found in connection with cases of chorea.

The social service division is one of the most important parts of the systematic care of cardiac children. As has already been stated, many factors of living and hygiene may be operative in the spread of rheumatic fever. It is the social worker who goes into the home to observe these conditions and whose duty it is to try and adjust difficulties whenever possible. Perhaps it is unnecessary to say that the social worker must be intelligent, possess common sense, and have had some training in public health problems. One social worker can adequately handle not more than one hundred patients.

Each clinic should have a clinic executive. She may be the chief social worker, or the nurse in charge of the clinic. She will see that the patients are enrolled, histories taken, and the temperature, pulse and weight determined, before the child is examined. She, or one of her assistants who may be a volunteer worker, will see that arrangements are made for special examinations such as x-ray, electrocardiograms, etc.

An appointment system is highly desirable. The physician should state definitely the return date for each patient, who is informed of this date. A postal card is sent to remind the patient of his appointment. If he does not return at this time, a second card is sent giving him an appointment for the next clinic session. If he does not respond then, a letter is sent, and if this fails to bring him back, the social worker should immediately make a house visit to determine the reason for the delinquency. The patient may be ill in another hospital, may have died, or moved, or be going to a private physician. If he is simply disgruntled with the clinic, the visit of the social worker is very likely to set matters straight. Whatever the circumstances, this visit will determine whether the child should be kept on as a member of the clinic or whether the responsibility of the clinic is ended.

The social worker should certainly keep up to date on the status of every patient, otherwise cases will be lost to the clinic. Obviously, such a system can be carried out only if the social service personnel is adequate in numbers. The clerical work involved in sending out cards and letters can be done by part-time workers who will notify the social service workers when a house visit is needed for follow-up. The time of the social service worker is much too valuable to be consumed in duties which can be performed by a less highly trained individual.

Any patient who has made no clinic visit for six months should be removed from the active list, but before doing this, every reasonable means should be taken to get the patient back in the clinic. The only facts which justify dropping a case are (1) death, (2) removal to another district, and (3) care by a private physician. Inadequacy of social service work is usually responsible for such reasons as "lack of coöperation." In a large city social service work is facilitated by limiting the use of the clinic to patients living within a reasonable distance from the clinic.

The physical equipment of the clinic should consist of a waiting-room and either cubicles or tables enclosed by screens for the examination of the patients. An x-ray plant must be available, and a fluoroscope

and electrocardiograph are desirable. There should be a sphygmomanometer, spirometer, ophthalmoscope, tongue depressors, and facilities for urinalysis and exercise tests. Other points, such as ventilation, adequate plumbing, desks for physicians and other workers, are similar to those required by any first-class clinic.

The matter of records is one which should be carefully considered by every clinic. Most physicians recognize the value of history and physical examination charts which cover adequately the type of case involved. Such charts have been designed by the Research Committee of the Heart Committee of the New York Tuberculosis and Health Association. These or similar records should be used by any cardiac clinic which wishes to have and maintain a high standard. The advantage of a graphic system for recording physical signs has been described in a previous chapter. It is highly desirable to have records kept in such a form, and so completely that anyone desiring to study a series of cases will be able to get the data easily and with fair assurance that they are accurate. In the case of a disease about which so much remains to be learned as rheumatic fever, the availability of complete records of cases for analysis is of great importance. This has been demonstrated by the paper of Wyckoff and Lingg, and of Wilson, *et al.*, who obtained their information from data recorded on the forms referred to.

COÖPERATING AGENCIES

Besides the care given the child in the cardiac clinic and by the cardiac clinic social worker, other agencies can be used. The school which a cardiac attends should know the child's physical condition, and if he is unable to take part in the gymnasium work, or march in line, or in fire drill, the school should be notified. Children in Classes 1 and 2a are able to attend a regular school. A child classified as 2b should not be in school because of the extent of his physical disability. The cardiac child should be encouraged to live as normal a life as he can within his limitations, and this is made possible only through the cooperation of everyone with whom the child has contact.

In an older child the problem of his future life is most important, and it is desirable both from the standpoint of his physical condition and psychological state to find an occupation which is consistent with his disability. Vocational guidance bureaus can be of great service in finding suitable training. Officials in high schools where different trades are taught can also be most helpful.

When the cardiac child is ill at home, care can be provided through a visiting nurse service which will give nursing care, instruct the mother, and see that there is adequate medical supervision of the case.

Convalescent Homes.—In many cases the most helpful thing that can be done for a child is to remove him from his usual environment. In New York City there are four convalescent homes for cardiac children, which cared for 538 cases during the year 1928. When this figure is compared with the number of cases enrolled in a single clinic (Bellevue Children's Clinic, about 400 cases) the need for more convalescent homes is apparent. Of the children in Class 1, 90.9 per cent improved in general condition during their stay in the homes; of those in Class 2a, 85.7 per cent improved, and of Class 2b, 78.6 per cent. Most of the children, therefore, are benefited by the care they receive in these homes, and if they could be kept for a period of years, perhaps even greater and more permanent good could be done. Children who are recovering from an attack of rheumatic fever or of heart-failure are eligible for convalescent care. If a child begins to lose weight or show any of the signs which point to the possible onset of a siege of rheumatic fever, the attack sometimes appears to be averted by sending him promptly to one of these homes.

Children are taught during their stay in convalescent homes how to lead well-ordered, hygienic lives, to eat well, rest sufficiently and acquire good health habits. They usually gain in weight. Sometimes this gain is lost on returning to the city, and sometimes their good habits are forgotten very soon, but a great many maintain the gain which they have made. The change in children who have done well in the convalescent home is often very striking and satisfactory. A child who has gone away thin, pale and listless may return with a gain of 10 pounds or more, have a good color and be full of energy. The cases which derive most benefit are usually those in Classes 1 and 2a since the extent of the cardiac damage is not as great as in the other classes and no active infection is present. On the other hand, surprising improvement occasionally takes place in the cases which belong in the 2b or 3 Class.

As has been stated before, the nutrition of these children is a most important factor in their progress. A child who can be made to gain weight has a brighter outlook than one whose weight remains stationary or is decreasing. The value of the convalescent home, aside from the advantage in removing the child from the environment which helped induce the illness, lies in the impetus given to improvement in

nutrition and hence greater power of resistance to further infection. The demand for more convalescent homes is insistent and from a superficial point of view the ideal might seem to be to have enough homes to take care of all cases for as long a period of time as seems advisable to attain maximum benefit. Such a condition would not, however, permanently solve the problem. The social class involved needs education in matters of hygiene, sanitation, standards of living and all matters pertaining to health and the prevention of disease. The social worker who teaches a mother how to care for her sick child, how to protect the well members of the family, and how to arrange her household in the wisest manner does a more far-reaching and constructive piece of work than the one who simply removes the child from the home. The task of education is a challenge to physicians, social workers and all health agencies. It demands a background of vision, patience and courage. Although the results may not be evident for many years, they are most certainly worth striving for. Each individual case must be cared for in the best possible way, but it must be reiterated that society as a whole is affected by this disease, and that the public health point of view must always be kept uppermost in mind.

APPENDIX

A NOMENCLATURE FOR CARDIAC DIAGNOSIS

PREPARED BY THE COMMITTEE ON RESEARCH OF THE AMERICAN HEART ASSOCIATION

From a study of the following tables it will be seen that a complete diagnosis is intended to include not only a statement of the structural changes in the heart, but also one concerning the cause of such changes, one indicating the type of disturbance of physiological function, and finally one defining the functional capacity of the heart.

Such a comprehensive diagnosis obviously requires careful consideration of every aspect of each case and is, therefore, essential to sound prognosis and effective treatment. Moreover, the general use of such a nomenclature and such complete diagnoses should aid greatly in the accumulation of statistics sufficiently comprehensive and accurate to be of real and permanent value.

Each diagnosis, to be complete, should include one or more titles from each of the groups A, B, C, D. Groups E and F, of course, require only reference to the etiological factor.

DIAGNOSIS OF HEART DISEASE

A. ETIOLOGICAL

1. Unknown
2. Rheumatic fever ¹
 - (a) Polyarthrititis
 - (b) Chorea
 - (c) Growing pains
 - (d) Tonsillitis
 - (e) Pharyngitis
 - (f) Others, as purpura, erythema nodosum, etc.
3. Syphilis ¹
4. Bacterial infection ¹ (*specify bacterium if possible. Here should be classified subacute infective endocarditis—i.e., Streptococcus viridans—chronic endocarditis, etc.*)
5. Thyroid ¹
 - (a) Hyperthyroidism
 - (b) Hypothyroidism

¹In the case of each of these conditions, it should be noted, if possible, whether the etiological factor is still active or is inactive.

6. Toxic
 - (a) Bacterial toxin
 - (b) Mineral
 - (c) Vegetable
 - (Specify if possible.)
7. Neurosis (cardiac)
8. General systemic disease (*specify the disease, e.g., arteriosclerosis, chronic nephritis, diabetes, emphysema, hypertension, anemia, etc.*)
9. Trauma
10. Congenital developmental defect

B. ANATOMICAL

1. Undiagnosed
2. Atrophy of heart
3. Enlargement of heart
4. Hypertrophy of heart
5. Dilatation of heart
6. Ventricular preponderance
 - (a) Right
 - (b) Left
7. Auricular hypertrophy
8. Cardiac thrombosis
9. Cardiac infarction
10. Rupture of heart
11. Myocarditis, acute
12. Myocarditis, chronic
13. Fibrosis of myocardium
14. Fatty infiltration
15. Fatty degeneration
16. Endocarditis
 - (a) Acute
 - (b) Chronic

(Include the continuous activity of a valvulitis, of papillary muscle, chorda tendineæ or mural infection. Usually 16 would precede the 17 diagnosis.)
17. Cardiac valvular disease
 - (a) Aortic insufficiency
 - (b) Aortic stenosis
 - (c) Mitral insufficiency
 - (d) Mitral stenosis
 - (e) Pulmonic insufficiency
 - (f) Pulmonic stenosis

- (g) Tricuspid insufficiency
- (h) Tricuspid stenosis
(Should be thought of as active or inactive as expressed under Etiology.)
- 18. Congenital abnormality (*specify lesion if possible*)
- 19. Pericarditis, acute
 - (a) Serous
 - (b) Serofibrinous
 - (c) Purulent
- 20. Hydropericardium
- 21. Hemopericardium
- 22. Pneumopericardium
- 23. Adherent pericardium
- 24. Aortitis
 - (a) Without dilatation
 - (b) With dilatation (aortitis, syphilis, in contradistinction to 29, which includes arteriosclerotic changes in the aorta)
- 25. Tumor of (*specify part affected*)
- 26. Aneurysm of (*specify affected vessel*)
- 27. Embolism of (*specify affected vessel*)
- 28. Thrombosis of (*specify affected vessel*)
- 29. Arteriosclerosis of (*specify locality*, as coronary arteries, cerebral arteries, etc.)
- 30. Arteritis of (except aorta) (*specify locality*)
- 31. Periarteritis of (*specify locality*)
- 32. No circulatory disease
- 33. No cardiac disease

C. PHYSIOLOGICAL

- 1. Regular sinus rhythm
- 2. Ectopic rhythms (without tachycardia) (This is to be differentiated from ectopic rhythm with tachycardia of the paroxysmal type. In the ectopic rhythms without tachycardia, it is usually believed that the new rhythm has developed because of a depression of the normal pacemaker. *Example*: Under this heading should be classified such a rhythm as nodal rhythm.)
- 3. Vagal arrhythmia
 - (a) Sinus arrhythmia
 - (b) Sino-auricular standstill
 - (c) Simple bradycardia
 - (d) Ventricular escape
 - (e) Wandering pacemaker

4. Tachycardia (not paroxysmal)
 - (a) Sinus
 - (b) Ventricular (those which have been reported showing this arrhythmia associated with digitalis poisoning should be classified here)
5. Premature contractions
 - (a) Auricular
 - (b) Junctional
 - (c) Ventricular
 - (d) Unknown
6. Paroxysmal tachycardia
 - (a) Auricular
 - (b) Junctional
 - (c) Ventricular
 - (d) Unknown
7. Auricular flutter
 - (a) Paroxysmal
 - (b) Chronic
8. Auricular fibrillation
 - (a) Paroxysmal
 - (b) Chronic
9. Ventricular fibrillation
10. Auriculoventricular heart-block
 - (a) Partial block
 - (1) Prolonged conduction time
 - (2) Occasional dropped beats (irregular)
 - (3) High-grade block (2:1; 3:1; etc.)
 - (b) Complete block
11. Intraventricular block
 - (a) Partial
 - (b) Bundle-branch, right or left
12. Pulsus alternans
13. Valvular incompetency
 - (a) Mitral incompetency
 - (b) Tricuspid incompetency
 - (c) Pulmonic incompetency
 - (d) Aortic incompetency
14. Hypertension
15. Congestive heart-failure
16. Effort syndrome
17. Anginal syndrome

D. FUNCTIONAL CAPACITY

1. Patients with organic heart disease, able to carry on ordinary physical activity without discomfort
2. Patients with organic heart disease, unable to carry on ordinary physical activity without discomfort
 - (a) Activity slightly limited
 - (b) Activity greatly limited
3. Patients with organic heart disease and with symptoms or signs of heart-failure when at rest, unable to carry on any physical activity without discomfort

E. POSSIBLE HEART DISEASE

Patients who show abnormal signs or symptoms referable to the heart, but in whom the diagnosis of heart disease is uncertain

F. POTENTIAL DISEASE

Patients without circulatory disease whom it is advisable to follow because of the presence or history of an etiological factor which might cause disease

EXAMPLES

Heart Disease: A. Active syphilis; B. Aortitis with dilatation, aneurysm of the ascending aorta; C. Regular sinus rhythm; D. Class 2a
or

Heart Disease: A. Inactive rheumatic fever (chorea); B. Valvular disease, mitral insufficiency and mitral stenosis; C. Auricular fibrillation; D. Class 2b

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